EXHIBIT 1

Expert Report of

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MetaMethod
South Lyon, MI

in the matter of

Hardwick v. 3M Company, et al.

December 14, 2020

MetaMethod

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Dominik D. Alexander, Ph.D., M.S.P.H.

December 14, 2020

RE: *Kevin D. Hardwick, et al. v. 3M Company, et al.*

Dear All,

I am a Principal Epidemiologist with MetaMethod. I have extensive experience in health research methodology, meta-analysis, and disease causation, particularly in the conceptualization, design, analysis, and interpretation of epidemiologic studies. I have published on a diverse range of topics and types of studies, including original epidemiologic research, qualitative reviews, systematic weight-of-evidence assessments, and quantitative meta-analyses. Because of my expertise in research methodology, I have served as principal investigator on numerous projects involving a wide variety of exposures and health outcomes. My research areas include but are not limited to: occupational and environmental exposures to substances, such as asbestos, benzene, trichloroethylene, solvents, pesticides, arsenic, perfluorinated compounds, and dioxin; community health studies and cluster investigations involving air, water, and soil exposures; clinical, pharmacoepidemiology, and medical device studies including clinical trial design and support. In addition, I have extensive experience in nutritional epidemiology and have conducted systematic reviews and meta-analyses of dietary and nutritional factors and cancer, cardiovascular disease, type 2 diabetes, hypertension, and body composition. My work in this area has involved studies of dietary patterns, intake of whole foods, and dietary supplements, such as meat and fat intake, dairy and egg consumption, breakfast eating, multivitamin and mineral supplements, fish oil, caffeine, coffee, and infant formula.

I have 210 peer-reviewed published articles, professional presentations, abstracts, and book chapters. I frequently present on the understanding and interpretation of epidemiologic evidence in a variety of professional venues, such as national conventions, scientific conferences, and governmental regulatory forums. I serve on the editorial boards of the American Journal of Clinical Nutrition, PLOS ONE, and Frontiers in Nutrition Methodology. I am a former Visiting Professor of Epidemiology at the University of Copenhagen in Copenhagen, Denmark, and am currently an Affiliate Associate Professor at Colorado State University. In addition, I regularly serve on scientific committees and scientific advisory meetings. I was awarded a National Cancer Institute Fellowship for Cancer Prevention and Control and was the 2010 recipient of the University of Alabama-Birmingham School of Public Health alumnus award for scientific excellence, based on recognition of my "significant scientific contributions through demonstrated commitment and exemplary leadership in empirical research, research methodology, or theory building or adaptation."

My CV, list of testimony, and rate sheet are included as attachments to this report.

Materials Reviewed

I have reviewed the following documents provided to me by counsel:

Source
Hardwick v. 3M Company, et al. – Class Action Amended Complaint
Plaintiff's Motion for Class Certification, July 31, 2020

In addition, I have reviewed the epidemiologic literature of all diseases, health outcomes, and clinical markers among PFASs-exposed study populations (see Appendix C).

Scope of Work

I have been retained by counsel for the Defendants in this case to examine the feasibility and scientific methodology to study a nationwide class whereby the members are allegedly at an increased risk of disease and health conditions as a result of exposures to one or more per- or polyfluoroalkyl substances ("PFAS"). In addition, I have critically examined the peer-reviewed epidemiologic literature of chronic and non-chronic diseases, health conditions, and clinical markers among PFAS-exposed study populations.

For the purpose of this report, I discuss the feasibility and scientific considerations of the Plaintiff's proposal to study a nationwide class of individuals exposed to PFAS. I also provide appendices that discuss an overview of the principles and practice of epidemiology, including study design differences, measures of association, internal and external validity parameters, and methodology to examine a body of epidemiologic literature (Appendix A); the various study population sources and their general PFAS exposure level ranges (Appendix B); and the state-of-the-epidemiologic science of human disease, health conditions, and clinical markers among PFAS-exposed study populations (Appendix C).

Feasibility and Scientific Methodology to Study a Nationwide Class

I have reviewed Plaintiff's Motion for Class Certification, focusing on the Plaintiff's proposed "program for study" and any proposed methodology to conduct such a study. Based on review of the Plaintiff's Motion for Class Certification, a program for study has been proposed based on:

> *A class of "any individual residing within the United States at the time of class certification for one year or more since 1977 with 0.05 parts per trillion (ppt) or more of PFOA and at least 0.05 ppt or more of any other PFAS in their blood serum."*

In the following sub-sections, I discuss why the proposed program for study is infeasible, methodologically limited, and scientifically improper.

1) No Study Design or Methodology

In the Motion for Class Certification, it is suggested that the classwide relief would lead to medical and scientific studies, testing, and analysis of all potential class members. However, no proposed study design, cohort enumeration strategy, sampling techniques, outcome validation, or statistical analysis methodology has been provided. In fact, no scientific methodology whatsoever has been proposed (aside from suggesting that a study similar to the Mid-Ohio Valley studies could conceivably be performed). Nor has a protocol for Institutional Review Board[1] approval been supplied.

The Plaintiff requests that the court oversee a program that includes epidemiologic studies focusing on the "causal connection(s) between any single or combination of PFAS in human blood and bodies and any injury, human disease, adverse human health impacts, or other health risks." However, the Plaintiff has provided no methodology to systematically evaluate the weight of the evidence to make causal inferences. He seemingly implies that the proposed study would be sufficient to make general causation inferences with respect to the thousands of different types of PFAS chemistries that exist. This is unscientific and does not follow principles of rigorous scientific methodology, which require, among other things, examination of the total body of literature in drawing causal conclusions, as I discuss in Appendix A.

2) The Proposed Study Cannot Answer Questions of Risk or Causation for the Entire Class

The Plaintiff alleges that "all" proposed class members face the "same persistent, continuing, and accumulating contamination," and implies they are all equally at risk of developing various diseases, including cancer. However, he erroneously suggests that any level of exposure increases the risk of disease. Even for substances that have been shown to cause human disease, volumes of scientific studies refute the notion that any non-zero level of exposure is sufficient to do so. Studies involving exposures to various types of PFAS do not support that notion either. Furthermore, the Plaintiff does not specify which diseases or conditions he alleges are associated with exposure to any PFAS, and he provides no scientific literature to support his allegations for any specific disease.

Dose-Response and Dose-Threshold Are Fundamental Properties of Causation

As indicated, the Plaintiff suggests that all class members (i.e., study participants) are exposed to some types of PFAS, and that everyone exposed (i.e., everyone in the class/study) is at increased risk of disease. As a result, the proposed study would essentially treat all of the class members the same for purposes of monitoring. There is no reliable scientific evidence to support this underlying

[1] An Institutional Review Board, or IRB, protects the rights and welfare of human subjects in research projects. IRB represents a designated group to review and monitor biomedical research involving human subjects. For example, see: https://www.fda.gov/regulatory-information/search-fda-guidance-documents/institutional-review-boards-frequently-asked-questions

assumption. One of the fundamental principles of epidemiology is the concept of dose-response, which means that for exposures that can cause adverse effects, the incidence of those effects is expected to increase among populations exposed to higher levels of the substance at issue. In other words, higher exposures should result in a greater risk of disease while lower exposures should have less risk. In fact, the presence or absence of a dose-response relationship in epidemiology studies is one of the critical factors considered in evaluating whether a body of scientific evidence indicates that a causal relationship exists between exposures and the effects being studied. A related concept is dose-threshold, which means that, even for exposures to substances that can cause adverse effects, there is an exposure level (a threshold) below which there is no increased risk of disease. Moreover, when different diseases can be caused by the same substance, those diseases often have different threshold dose levels. Most substances that are established as increasing the risk of disease exhibit a threshold behavior. Establishing a lack of threshold requires affirmative evidence of increased risks at all levels of exposure – it cannot reliably be assumed merely from a lack of evidence of a threshold. Here, in fact, the evidence actually demonstrates the opposite, that there are above-background PFAS exposure levels that do not lead to increased risks of disease. As summarized in Appendix C of this report, the epidemiology of PFAS fails to show a clear and consistent dose-response relationship for any human disease with any type of PFAS that has been studied, much less the existence of materially increased risks of adverse effects at all levels of exposures for the numerous types of PFAS compounds that have been studied. Given the lack of evidence ruling out a threshold dose, the proper epidemiologic method for determining whether and to what extent any particular person may be at increased risk from exposure to any particular PFAS requires, first, determining the exposure level of the individual to the particular PFAS along with other relevant individual factors and then, evaluating the available epidemiologic evidence of causation for populations with comparable levels of exposures and characteristics.

Assessment of Risk Requires Individualized Accounting of the Causal Factors of Disease

On a population basis, and certainly on an individual-by-individual basis, estimating disease risk attributable to an exposure requires consideration of all of the causal factors potentially at play for the individual/population. That is, erroneous conclusions can be drawn regarding the alleged exposure of interest (e.g., any particular PFAS) unless individualized factors that contribute to a person's disease or health symptoms are taken into account and analyzed. Indeed, a fundamental requirement for properly interpreting study results is assessing the extent to which the study accounted for alternate potential causes or risk factors (including preventive factors) of the outcome, and the quality of the methodology in doing so. Human disease and adverse health conditions are caused by, and/or exacerbated by, numerous factors that vary widely between individuals, including dietary and lifestyle behaviors (e.g., cigarette smoking, alcohol intake, diets high in certain fatty acids), medication use, genetics, family history of disease, comorbidity, and body mass index among numerous other factors. Many of these factors and their relationship with human disease have been evaluated in large volumes of analytical epidemiologic studies and are supported by clear dose-response patterns. In any given individual, the presence or absence of such factors may significantly impact the overall risk of a specific condition or several conditions, depending on the risk factor. If one were to assume the scientific evidence indicated that exposure to any PFAS did cause any human diseases/conditions (which it does not), the presence of these other factors in the proposed

population to be monitored would impact any determination of the risk that may be attributable to those exposures.

3) The C8 Science Panel Studies in the Mid-Ohio Valley Community Cannot be Used as a Model

The Plaintiff seemingly suggests using the C8 Science Panel studies among Mid-Ohio Valley community members as a model for the testing, monitoring, and research for the proposed nationwide PFAS exposure and health study. His reliance upon the methodology of the C8 Science Panel studies for the proposed nationwide study is flawed for several reasons:

- It is highly unlikely on both scientific and logistical grounds that the exposure characterization and dose-response analyses utilized in the C8 Science Panel studies would be possible in the proposed nationwide study. To begin, the sources, types, timing, and routes (e.g., air v. water v. food) of PFAS exposures at issue in the proposed nationwide study and C8 Science Panel studies are heterogeneous. The Mid-Ohio Valley study population involved known historical emissions of a single type of PFAS (PFOA) emanating from a single company's operating site and affecting public water suppliers. Much of the study population's PFOA exposures were thought to be attributable to this source and, based on this assumption, modelling was performed to attempt to estimate historical population exposures. A nationwide class would not have the same identifiable source or timing of PFAS exposures, and would have widely variable levels of exposure to a widely variable number of PFAS chemistries. In short, the exposure profiles of individual class members would be significantly different and highly variable and could involve up to thousands of different substances with widely varying toxicological properties. The levels and range of exposures present in the two study populations (i.e., the Mid-Ohio Valley study population and the proposed class comprising most of the U.S. population) are also vastly different (see Appendix B). Unlike the Mid-Ohio Valley study population, most class members would have only background levels of exposure to one or more PFAS chemistries from varied sources. Without reliable, consistent, representative, and above-background exposure profiles for study participants across a wide range of exposure levels, any meaningful or scientifically valid dose-response analyses would not be possible.
- Findings from the C8 Science Panel studies cannot be generalized to other populations. The C8 Science Panel applied a unique standard that had been negotiated in a class settlement and was neither a medical nor a scientific causation standard. The Mid-Ohio Valley study population was agreed to by the parties to the Leach case, as a compromise, and involved alleged water contamination by a single substance from a single site. One of the key factors when designing a scientifically valid study that is capable of being generalized is the enumeration of a study sample population that is representative of the broader population. There is no evidence to support the notion that creating a nationwide study population can be justified on the basis of the negotiated and unique resolution in the Leach class action.
- Plaintiff's apparent proposal to simultaneously study the potential health effects of each of the thousands of different types of PFAS would require a much more rigorous methodology

than that utilized by the C8 Science Panel in its studies. Indeed, doing so in a scientifically valid epidemiologic analysis requires, among other things, isolating the possible effects of each individual PFAS exposure by controlling for the potential effects of all other PFAS exposures. The C8 Science Panel studies evaluated a single compound, PFOA, not the thousands of different PFAS chemistries that are proposed to be studied by the Plaintiff. The C8 Science Panel studies did not (and were not able to) control for (or statistically adjust for) the numerous other PFAS to which the community members may have been exposed. Thus, the prior C8 Science Panel studies cannot serve as a model to validly analyze scientific data even for PFOA, much less for multiple other PFAS chemistries.

- The C8 Science Panel studies did not answer causation questions. Plaintiff contends that the original C8 Science Panel studies "provided an effective mechanism for resolving shared, common scientific questions regarding the causal connections between PFAS exposures and health risks to class members." This is fundamentally untrue on scientific grounds. In fact, the C8 Science Panel authors recently published a review on PFOA and health, and concluded that, "Overall, the epidemiologic evidence remains limited" (Steenland et al. 2020). In addition, the study designs utilized in many of the Mid-Ohio Valley studies were cross-sectional (or used cross-sectional methodology within a longitudinal analysis), and were neither designed nor equipped to address questions "regarding the causal connections between PFAS exposures and health risks" as suggested by the Plaintiff.

4) Exposure to Any PFAS has Not Been Established as a General Cause of Any Disease Outcome

Notably, review of the large body of epidemiologic analyses that have been performed to date (see the Reference List in Appendix C), including numerous studies conducted by the C8 Science Panel on which Plaintiff models his study proposal, indicates that the epidemiologic evidence does not support an independent association between exposure to any type of PFAS and an increased risk of human disease, health condition, or abnormal clinical marker (see Appendix C). Moreover, the epidemiology does not support a conclusion that exposure to any PFAS is a general cause of any disease or health condition. Furthermore, to my knowledge, no health organization, such as the National Cancer Institute, the American Cancer Society, the American Lung Association, or the American Heart Association, has ever concluded that any PFAS is an etiologic factor for any type of disease. Indeed, to the contrary, numerous regulatory agencies and expert panels that have assessed the potential relationship between a variety of different health effects and exposure to various PFAS types (with PFOA and PFOS being the most heavily studied) have not found causal associations for specific health conditions based on their review (ATSDR , NTP 2016, Australia PFAS Expert Health Panel 2018, ATSDR 2019, Michigan PFAS Science Advisory Panel 2018, EPA 2016b, a)[2]. This is important for assessing the propriety of Plaintiff's proposal here. Given the need to evaluate

[2] The European Food Safety Authority's (EFSA) Panel on Contaminants in the Food Chain suggested that there is some support for causality for some outcomes and/or clinical markers for PFOA and PFOS. However, the panel noted several limitations in their conclusions, such as confounding, excretion that affects circulating concentrations of PFOA and PFOS, lack of clinical relevance, and co-exposures to a broad range of compounds (Knutsen et al. 2018).

causation in the context of the larger body of available scientific evidence and the lack of evidence of causation for the particular PFAS that have been studied to date, Plaintiff's proposed nationwide study program in and of itself would not and could not provide the answers to the causation questions he claims are needed.

Diseases and Health Outcomes

Studies involving the highest exposed populations (i.e., PFOA and PFOS exposed workers in the occupational cohorts) do not support a causal inference that exposures to either of these substances cause any human disease or health condition. Many associations within and across studies are inconsistent, suggest that there are inverse associations, lack statistical significance, or show only minimal effects (if any). Most of the occupational studies are longitudinal, thereby allowing researchers to evaluate the capacity of exposure to particular PFAS to predict the risk of incidence of disease or of deaths due to specific outcomes. Furthermore, the exposure levels in the occupational cohort studies are considerably higher (sometimes by an order of magnitude or more) than most other studies, such as those that utilize data from NHANES (general population) (see Appendix B). Given the lack of consistent associations observed in these cohorts, populations exposed at much lower levels of particular PFAS, such as the class proposed by Plaintiff, would not be expected to be at increased risk of adverse health outcomes. Any outcome of Plaintiff's proposed study involving these particular PFAS would have to be interpreted in light of these findings and thus could not, of itself, establish causation for the entire proposed class.

Inconsistent associations – some showing increased risks while others indicate decreased risks or no associations at all -- and sporadic observations of statistically significant relative risks[3] (that have not been replicated) have been reported across community studies involving significant above-background exposures (e.g., C8 Science Panel studies of the mid-Ohio Valley residents). While some potential positive associations have been observed for some outcomes in some community studies, the findings are often inconsistent both within these studies and between studies. For example, testicular cancer incidence was increased among Little Hocking residents but was *decreased* among residents in the other water districts who had exposure levels above background but below the levels found in Little Hocking. Importantly, the epidemiologic evidence also was not supportive of an increased risk of testicular cancer in PFOA exposed workers, even though the exposure levels for these workers were higher.

Associations from study populations exposed to essentially background PFAS exposure levels (e.g., NHANES data analyses) are inconsistent, and many of these studies suffer from methodological and analytical limitations (see Appendix A). These limitations include, among many others: 1) utilization of cross-sectional studies that are not designed to evaluate temporal relationships, as prevalent outcomes or current conditions are ascertained simultaneously with single measurements of exposure, 2) limited control and adjustment of confounding factors, including not accounting for changes in relevant confounding factors over time, 3) analyses of study populations with narrow ranges of low exposure levels, and 4) failure to account for the many statistical comparisons

[3] See Appendix A for a description of measures of association, such as relative risk, and statistical significance.

performed that can increase the odds of chance findings. In some studies, potential positive associations are observed, but either no associations or inverse associations are reported for that outcome in more highly exposed study populations. Tellingly, there is no consistency across studies, despite decades of extensive study of PFOA and PFOS.

Plaintiff's proposal to conduct yet further studies of these chemistries on background-exposed populations will not meaningfully add to the epidemiologic database or resolve these inconsistencies. Because of the difficulties of determining past exposures and past disease/health statuses for a heterogeneous nationwide study population that would allow following any such study population over time (longitudinally), descriptive cross-sectional and ecologic designs are the types of study designs that would likely be utilized for any attempted nationwide class study proposed by the Plaintiff. These types of study designs have significant limitations, including an inability to establish causation. A recent commentary authored by the former C8 Science Panel members (summarized below) concluded that "[a]dditional cross-sectional studies of low exposed populations may be less informative" (Steenland et al. 2020). Furthermore, the authors commented on studies of the general population, similar to the proposed nationwide class study, as follows: "Exploiting readily available data from general population studies is efficient but when focused on background exposures as reflected in biomarkers, differences may be strongly influenced by variability in uptake and excretion (rendering them vulnerable to confounding and reverse causation) and precluding historical exposure reconstruction which is feasible only if there is historical information on specific dominant exposure sources."

There are volumes of literature on studies of PFAS exposure that have utilized more rigorous analytical epidemiologic methods (as summarized below). Thus, the Plaintiff's motion to certify and study a nationwide class would be unlikely to advance the science. Furthermore, many if not most chronic disease conditions have latency periods that can range between 10 and 40 years, and any proposed study to evaluate the potential risk of disease among PFAS-exposed study populations must incorporate longitudinal and temporal methodology, such as reconstructing historical exposure data for each and all of the separate types of PFAS exposure and resulting blood levels for the entire study population (this is infeasible for a nationwide class) and/or following the entire study population prospectively for several years or decades, but with repeated measures of serum levels and repeated measures of potential confounding and effect modifying factors.

Clinical Markers[4]

Whether analyzing exposure data and clinical markers in a single study or interpreting findings across the body of literature, some crucial methodological and analytical factors must be considered when interpreting the evidence. Some, but not all, of these important factors are:

i. An individual's level of any particular clinical marker often varies considerably, and serum measurements may follow a normal within-person distribution and fluctuate on a

[4] A biological measure that may indicate normal or abnormal physiological functioning, including the presence of disease or health condition. Note: an abnormal laboratory value may not indicate a current disease state.

regular basis. For example, measurements may vary by time of day or over time. Thus, the collection of a single measurement of one of these levels at one point in time may not be informative and repeated testing may be necessary. Furthermore, individual clinical markers, such as enzyme levels and serum measurements, need to be confirmed and validated, and such measurements need to be replicated over various time points.

ii. Even when a lab value is outside the generally accepted normal range, it does not mean it is indicative of a specific condition or disease.

iii. A single value at a specific point in time may not predict the magnitude or level of a subsequent test, nor does it necessarily predict an outcome.

iv. Tests should be considered collectively, and a single result in isolation may not be an indicator of dysfunction.

v. Studies of clinical parameters often report well over 100 results. Thus, due to the multiple comparisons made on the data, many findings *are expected* to be statistically significantly positive as a result of chance.

vi. Statistical significance does not equate to clinical significance. Even if a statistically significant difference for a marker value is observed, as shown in volumes of studies, the values typically remain in the normal range and are not indicative of an adverse health condition.

vii. Innumerable factors can affect the clinical marker levels. These include lifestyle factors, such as body weight, physical activity, smoking, alcohol use, and dietary habits; demographic characteristics, such as socioeconomic status, gender, and race; occupational factors, such as exposure to workplace chemicals; clinical factors, such as medication use and co-morbid conditions; and genetic factors, such as family history of disease and having certain syndromes. All of these factors would have to be considered on an outcome-by-outcome basis before meaningful conclusions are drawn.

viii. Furthermore, many of the results (correlations) for the clinical markers may be of undetermined clinical relevance, meaning that even if a serum parameter is elevated or decreased it may not be related to a specific condition or it may reflect an unknown array of conditions.

For example, several key medical surveillance studies of clinical health parameters are not supportive of an association between exposure to any PFAS and adverse disease outcomes that are correlated with the clinical markers of interest. Costa (2009) reported on health outcomes of workers in a PFOA production plant as part of a medical surveillance program. Workers in this population had very high PFOA exposure levels (an order of magnitude higher than many other study populations). Although PFOA serum levels were correlated with increased total cholesterol and uric acid (but within the normal range for uric acid), no significant adverse health outcomes were reported. This led the authors to conclude that "neither clinical evidence of specific disturbances or health disorders have been recorded over 30 years of medical observations of workers exposed to PFOA, having serum levels ranging from 0.20 to 91.9 μg/mL" (Costa, Sartori, and Consonni 2009). In a 2006 publication of community exposure to PFOA, Emmett et al. (2006) evaluated various health parameters among residents in the Little Hocking Water Association district, which has the highest exposure levels of any of the community studies. The median PFOA serum in these residents was 354 ng/mL (which can be converted to 0.354 μg/mL for comparison to the workers evaluated by

Costa 2009). The authors concluded that "No toxicity from PFOA was demonstrated using the measured endpoints…" (Emmett et al. 2006).

Furthermore, among the *analytical* epidemiologic studies involving *highly exposed* populations that have been conducted regarding clinical markers and serum values, there is no clear or consistent evidence that exposure to any type of PFAS results in clinically relevant changes in clinical measurements. Importantly, the analytical epidemiologic evidence does not support a conclusion that exposure to any PFAS causes an increased risk of the disease outcomes that are correlated with the clinical markers. For example, while certain studies show correlations between PFOA and higher cholesterol levels, no association for cholesterol was observed in a longitudinal incidence cohort study of DuPont workers with high exposure to PFOA (Steenland, Zhao, and Winquist 2015). If there was truly a relationship, one would expect to see it in the more highly exposed population. Furthermore, the epidemiologic evidence does not support an increased risk of cardiovascular or cerebrovascular disease among PFOA exposed study populations (see Appendix C). Because cholesterol is highly correlated with heart disease risk, it would be expected that if PFOA modifies cholesterol levels in a clinically meaningful way, heart disease would be elevated among the study populations with the highest exposures. This is not the case.

As with the proposed nationwide study of disease and health outcomes associated with particular PFAS types, any proposed study of clinical markers of a nationwide class would likely also involve a cross-sectional study design methodology. However, such a study design has significant limitations, including the inability to assess exposures and clinical markers across time in the same individuals (longitudinally) or to provide the scientific evidence of causality that the Plaintiff claims is needed. Under the factors noted above, such a study design would be methodologically unstable and could not ascertain, validate, and analyze longitudinal clinical marker data using proper epidemiologic study design techniques.

C-8 Science Panel Commentaries

Plaintiff models his proposal here on the C8 Science Panel studies. However, as noted below, after numerous studies on a variety of health endpoints across a large population with common exposures to one type of PFAS (PFOA), the evidence from these studies remains "limited" and fails to establish causation for any studied diseases, clinical markers, or health outcomes. Moreover, further studies on other populations continue to modify, and in some cases undermine, any import of the C8 study findings. Plaintiff's proposed nationwide study program would fare no better, is ultimately infeasible, and is even less likely to yield scientifically meaningful results.

As discussed briefly above, the C8 Science Panel was created out of the settlement terms of a class action lawsuit brought by members of an Ohio River Valley community for contamination of the water supply with C8 (PFOA). A "probable link," as that term was used by the C8 Science Panel, was the standard mandated by the settlement terms and is not synonymous with and is different from establishing "causation"[5]. A "probable link" is defined by the C8 Science Panel as "given the

[5] C8 Class Counsel Letters, dated 8/2/05, 1/22/10, and 1/24/10. Jack W. Leach, et al. v. E.I. duPont de Nemours and Company. Circuit Court of Wood County, West Virginia, Civil Action No. 01-C-608 Science Panel

available scientific evidence, it is more likely than not that among class members [in the underlying West Virginia class action lawsuit] a connection exists between PFOA exposure and a particular human disease." Importantly, the "probable link" standard is unique, and *not* an established scientific or epidemiological term for addressing disease causation.

In 2010, the panel members published a commentary on the *Epidemiologic Evidence on the Health Effects of Perfluorooctanoic Acid (PFOA)*, and concluded that the "epidemiologic evidence remains limited, and to date data are insufficient to draw firm conclusions regarding the role of PFOA for any of the diseases of concern" (Steenland, Fletcher, and Savitz 2010). However, the panel subsequently concluded that there was a "probable link" between PFOA exposure and hyperlipidemia, ulcerative colitis, thyroid disease, testicular cancer, kidney cancer, and pregnancy-induced hypertension.

The former Science Panel members, in addition to other authors, recently published a new commentary, *Review: Evolution of evidence on PFOA and health following the assessments of the C8 Science Panel*. The panel updated their review of the health effects of PFOA based on literature published through March 2020. The authors stated that this review "does not revisit the probable link decisions, but restates them, and then considers more recent evidence, including for some outcomes for which the Science Panel did not determine that there was a link to PFOA." The panel and other authors ultimately concluded, "Overall, the epidemiologic evidence remains limited" (Steenland et al. 2020). In fact, the panel has tempered their conclusions and appears to have downgraded the level of evidence to support probable links for several outcomes, while raising the possibility of an association with a couple of new clinical factors.

Probable Link Outcome	**Conclusions in Steenland 2020**
Kidney cancer	The epidemiologic evidence "remains supportive but not definitive." The evidence "remains suggestive although not consistent in newer studies."
Testicular cancer	The epidemiologic evidence "remains supportive but not definitive."
Pregnancy induced hypertension	"There are few subsequent studies with mixed results." There has been "little additional research to confirm or refute the original assessment."
Thyroid disease	The evidence is "suggestive but uneven." The evidence since the original Science Panel findings "has gotten weaker." There is "little consistency across studies so evidence for a causal impact on thyroid hormones remains weak."
High cholesterol	There is "consistent evidence of a positive association between PFOA and cholesterol, but no evidence of an association with heart disease." "The positive association could reflect confounding" and "[s]ome observations lend support for this view."
Ulcerative colitis	The evidence "still supports an association of PFOA with ulcerative colitis" although "the latest study from Sweden did not find a positive association." "Given the sparse literature, more studies are clearly needed to reach more definitive conclusions."
Additional Outcomes	**Conclusions in Steenland 2020**
Liver cancer	There is "little evidence for a relationship of PFOA."
Pancreatic cancer	There is "little evidence for a relationship of PFOA."
Prostate cancer	There is "some suggestive evidence… but results are inconsistent."
Heart disease	There is "no evidence of an association with heart disease."
Auto-immune diseases	There is "evidence for an association with ulcerative colitis, but not for other auto-immune diseases."

Kidney disease	The evidence is "suggestive but uneven." There is "little evidence of an association between PFOA and chronic kidney disease."
Liver enzymes	There is "evidence of an association with liver enzymes, but not with liver disease." "PFOA was positively associated with ALT levels above the reference range, although the clinical relevance of this elevation in enzyme levels is not clear."
Liver disease	There is "evidence of an association with liver enzymes, but not with liver disease." The data continues to support "no probable link."
Neurotoxicity	There is "no consistent evidence of increased risk." There is "no consistent evidence of an association between prenatal and/or early childhood PFOA exposure and neurobehavioral development."
Birth weight	Reductions in birth weight "may be due to reverse causality and/or confounding."
Fecundability	There is "little support for a consistent or substantial effect on fecundability."
Miscarriage	The associations have been "consistently null, indicating the absence of a discernible impact."
Asthma	The authors indicated that, "recent studies provide little support for an association between PFOA exposure and asthma or atopic dermatitis among children."
Atopic dermatitis	The authors indicated that, "recent studies provide little support for an association between PFOA exposure and asthma or atopic dermatitis among children."
Childhood infection	"Results of associations between PFOA exposure and childhood infection are mixed."
Infectious disease	There is "uneven evidence for an association." Evidence is "inconsistent."

5) Inability to Evaluate Synergy Between or Among Individual PFASs

The Plaintiff suggests that "some risks are known (such as those related to PFOA)" but additional study is necessary to understand the "synergistic effects of exposure to multiple types of PFAS." First of all, the scientific evidence has *not* consistently or reliably established known health risks for PFOA, and as indicated above, the former C8 Science Panel members recently concluded that the "evidence remains limited" and that, "it is disappointing that we do not have greater clarity on adverse human health effects associated with PFOA." Second, the Mid-Ohio Valley studies did not address any potential "synergistic effects" to various PFASs, nor did they even statistically adjust for the potential confounding influence of other PFASs, and they certainly did not address potential effect modification by PFASs through stratified analyses. Thus, the C8 Science Panel studies of the Mid-Ohio Valley community cannot serve as a model to evaluate any potential synergistic effects. Third, in his Motion for Class Certification (pg. 30-31), the Plaintiff seemingly suggests that it is scientifically justified to group PFASs together to draw conclusions on potential health effects. This is counter to his proposal to study "synergistic effects of exposure to multiple types of PFAS." The concepts of specificity and independence of association are important considerations when making causal inferences. Thus, grouping PFASs together may mask relevant associations for specific compounds should they exist. In addition, analyzing multiple PFASs in a statistical model may introduce multicollinearity,[6] which would ultimately produce unstable risk estimates, and analyzing numerous PFASs can result in spurious statistically significant associations from multiple comparisons. Finally, the Plaintiff offers no scientific methodology as to how he would propose

[6] Multicollinearity can occur when highly correlated variables are analyzed together in a statistical model, which may render the results unstable and discordant.

evaluating any potential independent effects, synergistic effects, multi-collinear associations, or spurious associations resulting from multiple comparisons.

6) A Study of a Nationwide Class Will Not Advance the Science

The Plaintiff is requesting scientific studies to support the claim that PFAS endangers the "world at large" because he assumes they can cause human disease. However, the Plaintiff fails to acknowledge the fact that a large volume of epidemiologic studies has been conducted on worldwide study populations with background, above background, and high levels of exposures to many different types of PFAS. There have been hundreds of published studies on PFAS exposure and risk of human disease, health conditions, and clinical markers, as described in Appendices B and C. The studies have included well-conducted analytical epidemiologic studies with decades of objective worker exposure data, occupational surveillance studies, above-background community exposure studies, nationwide cross-sectional and ecological analyses of the general population, and cross-sectional and ecological analyses of unique study population samples. Importantly, this topic area is under continual and frequent investigation, as new epidemiologic studies are published on a weekly to monthly basis. Furthermore, some large-scale multi-site PFAS studies are underway[7]. For example, the Centers for Disease Control and Prevention (CDC) and Agency for Toxic Substances and Disease Registry (ATSDR) began a multi-site health study "to investigate the relationship between drinking water contaminated with per- and polyfluoroalkyl substances (PFAS) and health outcomes." Their stated goal was "to understand the relationship between PFAS exposure and health outcomes in differing populations." The CDC/ATSDR study has systematically identified geographic areas to study the potential relationship between gradations of PFAS exposure and risk of adverse health outcomes. It is noteworthy that they documented specific endpoints to evaluate, procedures to enumerate the cohort, and methodology to ascertain exposure information. In stark contrast, the Plaintiff here provides *no* cohort enumeration strategies nor study methodology. Furthermore, he provides no information as to whether or how his proposed study would be able to (or could be able to) advance the science on this topic above and beyond the hundreds of published studies, including the current ongoing studies.

The Plaintiff offers no alternative types of studies that could be conducted to evaluate the potential relationship between exposure to any type of PFAS and disease. In other words, he offers no design to evaluate this issue, and he certainly does not propose any other type of study that has not already been conducted. Nationwide studies have been conducted. For example, there are, at minimum, dozens if not hundreds of studies of representative samples of the U.S. general population (i.e., NHANES), and these studies have not been informative for addressing matters of general causation. Indeed, even the C8 Science Panel members concluded that, "Additional cross-sectional studies of low exposed populations may be less informative" (Steenland 2020). Regarding clinical markers, "we believe that caution should be exercised about such studies because of the potential for reverse causality or uncontrolled confounding, which can be particularly important at low exposure levels where the exposure contrasts are modest, and more strongly influenced by complex behaviors such

[7] https://www.cdc.gov/media/releases/2019/p0923-cdc-atsdr-award-pfas-study.html

as diet." The proposed study to evaluate a nationwide class would likely be cross-sectional or ecologic (no alternative design has been proposed, and any alternative design would likely be even more infeasible), involve non-detectable to background levels of exposure, be unable to examine potential cause-and-effect relationships (reverse causality issues), and be limited in its ability to collect information on -- and control for -- confounding factors or effect modifiers. In short, no scientifically valid information would result.

7) The Plaintiff's Proposed Program of Study for a Nationwide Class Does Not Meet the Scientific Standards for Medical Monitoring

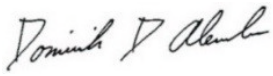
Putting aside the fact that the Plaintiff has not proposed a study design, cohort enumeration strategy, sampling techniques, outcome validation, statistical analysis methodology, or patient confidentiality procedures, such as an IRB-approved protocol, he also has not met the criteria for determining the appropriateness of a medical monitoring program[8]. The ATSDR has established scientific criteria to determine "when medical monitoring is an appropriate health activity and the requirements for establishing a medical monitoring program." The consideration of valid epidemiologic evidence is an essential component to medical monitoring. The ATSDR suggests that medical monitoring is not warranted until there are analytical epidemiology studies showing the causal nature of associations between exposures and outcomes. Here, however, volumes of epidemiologic literature do *not* support a causal relationship between exposure to any PFAS and human disease or adverse health conditions. Indeed, the agency indicates that, "In cases in which there is no known association between the exposure and specific adverse health effects (which could include health outcomes, illnesses, or markers of effect), medical monitoring is not an appropriate public health activity." Further, it states that before a medical monitoring regimen can even be considered, there must first be a "community identified" as being at "a significant increased risk of disease." There are three scientifically relevant factors in this statement that invalidate the Plaintiff's proposed study: 1) the Plaintiff does not propose to study a "community", rather, he seemingly proposes to study an entire nation; 2) he has not "identified" a target population at supposed increased risk; and 3) the epidemiologic evidence does not support a conclusion that a population (including an unidentified population) is at a "significant increased risk of disease." The ATSDR also indicates that, "The monitoring should be established for specific adverse health effects." The Plaintiff has proposed no specific outcomes to study.

I reserve the right to review any additional materials that become available in this matter and to amend my opinions accordingly.

Subject to the aforementioned reservation, this report contains a complete statement of all opinions I will express in relation to Plaintiff's motion for class certification in this matter and the basis and reasons for them, as well as the facts or data I considered in forming these opinions. I declare under

[8] https://www.govinfo.gov/content/pkg/FR-1995-07-28/pdf/95-18578.pdf

penalty of perjury under the laws of the United States that the foregoing is true and accurate to the best of my knowledge.



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Dominik D. Alexander, Ph.D., M.S.P.H.

December 14, 2020

RE: *Kevin D. Hardwick, et al. v. 3M Company, et al.*

Appendix A: General Principles of Epidemiology

The science of epidemiology involves investigating the distribution and determinants of diseases in human populations. Epidemiology is a tool for evaluating whether a given exposure is a cause of a specific disease or health outcome in humans, as well as establishing quantitative exposure-response relationships between a factor and the risk of disease.

- A principal focus of epidemiology is study methodology. Epidemiologists are trained to develop and design rigorous studies to evaluate the relationship between exposures and disease outcomes.

- In addition, some epidemiologists have unique expertise in synthesizing and summarizing a complex body of literature through meta-analysis methodology or weight-of-evidence assessments to make a scientific conclusion.

Well-conducted epidemiologic studies play an important (and necessary) role in assessing patterns of relative risk and general causal relationships between exposures, such as smoking, and outcomes, such as cardiovascular disease or lung cancer. In fact, epidemiology is the foundational scientific discipline that evaluates and estimates relative risk of disease among study populations (comprised of individuals), whether exposed to a chemical, consuming food or beverages, engaging in vigorous physical activity, or residing in a certain geographic location, among other factors.

Epidemiologic Measures

To measure the frequency of occurrence of health outcomes or disease in a population, epidemiologists may report the number of events per unit of population or time:

Incidence rate is the rate at which new events occur in a population, calculated as the number of new cases that occur during a defined period of time, divided by the average number of persons at risk of the disease during that period. For example, if 100 people develop a disease in an at-risk population of 10,000 persons during a 10-year period, then the incidence rate is 100 cases divided by (10,000 persons × 10 years) = 100 per 100,000 person-years or 1 per 1,000 person-years.

Mortality rate is the number of deaths, either overall or from a specific cause, identified in a given population during a defined period of time, divided by the average number of living persons in that population and time period.

Disease **prevalence** is the proportion of people in a population who have a certain disease or health-related events at a given time, calculated as the number of cases of a disease at a specific time or period, divided by the total number of persons at risk of the disease at that time or midway

through the time period. For example, if 20 people in a population of 10,000 persons currently have a disease at a specific point in time, then the point prevalence at that time is 20 per 10,000 persons or 1 per 500 persons.

Disease **incidence** measures the occurrence of new cases of a disease or health-related events. Prevalence measures the existence of both new and pre-existing cases of a disease or event. That is, incidence measures the risk of acquiring a disease or the occurrence of an event, and prevalence measures the risk of having a disease or an event. Thus, incidence data are preferable over prevalence data in analytical epidemiologic studies that aim to identify the relative risk of disease or the rate of the occurrence of an event based on a prior exposure or to evaluate potential causal risk factors. Prevalence is influenced by both disease incidence and disease duration (i.e., survival with or recovery from disease). For example, in cross-sectional studies (discussed below), the exposure and the outcome are assessed contemporaneously, which may limit their ability to determine whether the exposure actually increased the risk of disease (or ultimately caused the outcome). Of note, these are the study designs that the plaintiffs' experts in this matter primarily rely upon to draw their conclusions.

The Role of Epidemiology in Assessing Relative Risk and Disease Causation

The totality of evidence across all analytical epidemiologic studies can be synthesized and summarized, along with other available scientific evidence, to formulate an opinion on whether the exposure is: 1) associated with the outcome (typically upon review of patterns of relative risk estimates), 2) a clear risk factor for the outcome (by systematically accounting for and/or possibly ruling out the impact of chance, bias, and confounding, which is a type of bias), and ultimately 3) whether the exposure is a general cause of the disease (after application of well-established guidelines for causal assessments, such as those posited by Sir Austin Bradford Hill, 1965) (Hill 1965). Specifically, epidemiologists evaluate patterns of relative risk and associations that may exist in the presence or absence of bias and confounding. If an association exists between the exposure and outcome in a setting in which the potential for bias and confounding have been minimized, epidemiologists then examine whether the association is statistically significant as well as the strength of the statistical relationship. Provided adequate and reliable exposure data are available, epidemiologists evaluate potential dose-response patterns to determine whether risk is increased or decreased based on higher or lower levels of exposure. Importantly, the consistency of findings within and across studies are scrutinized, and any sources of design heterogeneity (between study variability) are examined.

There are two general types of epidemiologic studies. These are descriptive and analytical, and both designs are used for different scientific purposes.

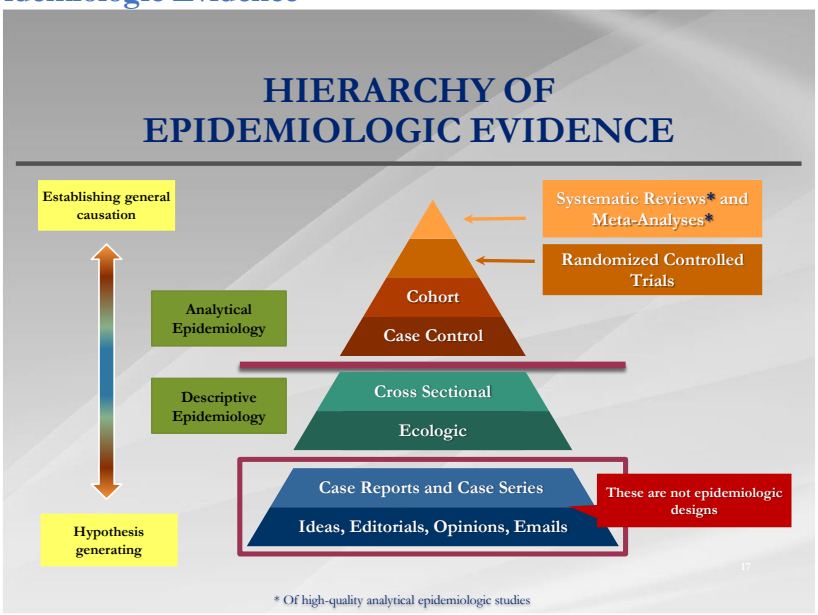
Descriptive studies, which are most often conducted on data from disease and mortality registries maintained by public health departments and government agencies, are generally used as surveillance tools to monitor the temporal trends and spatial distribution of disease occurrence. Such studies can alert the public to a concern if either the temporal trends or spatial distribution of the disease under consideration show unusual or unexpected patterns ***but cannot establish causal associations between a factor or exposure and the disease or event of interest***. An example of a descriptive epidemiological study would be a surveillance registry for birth defects at a population level. Descriptive epidemiology commonly includes surveys, which may be used to provide a 'snapshot' of

disease prevalence in a selected population group. Such designs lack the ability to determine cause and effect relationships because the exposure and outcome are typically ascertained at the same point in time. Descriptive studies are often limited in terms of ascertaining and analyzing critical information, such as data on potential confounding factors. Finally, it is difficult to account for the role of systematic bias in descriptive epidemiology.

Analytical studies, on the other hand, are specifically designed to estimate associations between exposures and disease or health outcomes. That is, they are designed to produce relative risk associations by comparing the morbidity or mortality experience (or health-related events) among exposed populations to the experience among lower or non-exposed populations. By nature of their analytical design, they are *testing* hypotheses. Analytical studies' use of comparison groups with statistical testing is what enables epidemiologists to evaluate patterns of relative risk. Importantly, such studies may be equipped to account for confounding factors, including the ascertainment of covariates (variables included in analytical models) of exposure and outcome, and allow for the control of the impact due to confounding. For example, analytical studies can control for confounding by including covariate factors in complex statistical models, such as regression methods or proportional hazards analyses, when estimating the association between the primary exposure of interest and health outcomes. In addition, analytical epidemiologic designs facilitate the assessment of many types of potential systematic bias by means of subject selection criteria, exposure and outcome information ascertainment and validation, analytical methodology, statistical significance testing, and results interpretation. Analytical study designs may include randomized controlled clinical trials, prospective or retrospective cohort studies, nested case-control studies, case-cohort studies, or case-control studies. However, even when analytical epidemiology study designs are used, there may be components of cross-sectional exposure estimates.

As illustrated below, there is a delineation between studies designed to 'test' hypotheses (i.e., analytical studies) and those used for surveys or surveillance that provide 'snapshots' of prevalence (i.e., descriptive studies, which are lower on the hierarchy of epidemiological evidence). Considerably lower on the hierarchy of epidemiologic evidence are case reports, case series, editorials, and written opinions. These are not considered studies *per se* because they are not designed to evaluate associations between exposures and outcomes, nor are they designed to provide an overview of data.

Hierarchy of Epidemiologic Evidence



Case reports are not studies and should not be utilized on an epidemiological basis to evaluate the potential relationship between exposures and health outcomes, particularly in the presence of available analytical epidemiologic studies.

- Case reports do not provide any methodological basis for determining general causation. It is important to differentiate between the epidemiological studies shown above and case reports.

- As described in the *Dictionary of Epidemiology*, case reports are regarded in the scientific community as "anecdotal evidence" that is "derived from descriptions of cases or events rather than systematically collected data that can be submitted to statistical tests" (Last 2001). Case reports may be used to generate hypotheses, but they cannot be relied upon for causal inferences.

- Case reports cannot provide evidence of an association. An independent association is a statistical relationship between two variables that is not due to chance, bias, or confounding. Since a case report contains no information on how frequently the disease occurs in the presence of exposure (except for the single occurrence of the case report) and no information on how often the disease occurs in the absence of exposure, there is no way to know whether there is any association between the two events.
- Tests of statistical significance cannot be performed on case reports. Thus, there is no valid or formal way to assess the probability that the two events (exposure and outcome) could have occurred by chance alone when in fact there was no true association between them. As a result, case reports are inherently unreliable, and provide no way of evaluating internal validity.

- Before making a determination of causation, epidemiologists must use proper and rigorously exercised methodology to test whether an association exists and whether the association may

be influenced by methodological error (i.e., systematic or random bias), distorted by extraneous factors (i.e., confounders), or explained by chance. Case reports are not designed to evaluate these critical and necessary methodological factors.

- Case series have the same limitations as case reports. A case series is a collection of patients with common characteristics, such as a common disease. In a case series there is no comparison group, and the selection of the cases is often based on a sample of convenience.

- The publication of case reports and cases series are inherently biased. Clinicians selectively report their observations and journals/editors may selectively publish such observations. These issues are referred to as reporting bias and publication bias, respectively. That is, they may lead to the perception of an association when one does not truly exist. In other words, a false-positive relationship.

- Case reports and case series may play a role in scientific inquiry, such as initially identifying an unusual disease or exposure in a patient that may be worthy of scientific investigation. However, addressing such an inquiry requires scientific evidence and valid scientific methodology that assesses whether there is an independent association in which the role of chance, bias, and confounding can be reasonably excluded as explanations. Case reports and case series cannot provide this evidence.

- For all of these reasons, case reports and case series are not designed to estimate associations nor are they considered reliable bases for making causal inferences. For example, the International Agency for Research on Cancer (IARC) indicates that case reports and case series usually suffer from incomplete case ascertainment in any defined population, lack definition and enumeration of the population at risk, and cannot estimate the expected number of cases in the absence of exposure. These uncertainties make case reports and case series inadequate to form the sole basis for inferring a causal relationship.

Types of Observational Epidemiologic Studies

Epidemiology is largely an observational discipline, with less control over exposures than human experimental or clinical studies. The two most commonly used types of analytical observational studies in epidemiology are cohort studies and case-control studies. Other, less informative study designs include cross-sectional and ecologic studies, which may be considered descriptive epidemiologic designs. Finally, experimental human studies, such as randomized controlled trials, may occasionally be used.

Cohort studies are analytical epidemiologic studies in which defined populations with various levels of exposure are observed over time to compare disease incidence or mortality rates across different exposures. Cohort studies may be prospective, meaning that exposure information is collected in the present, prior to disease onset, and participants are followed forward in time. Alternatively, cohort studies may be retrospective, meaning that past exposure information is attempted to be reconstructed based on existing information for participants, some or all of whom have already developed the disease(s) of interest. Prospective cohort studies are often analyzed using a statistical

approach that evaluates the occurrence of disease or health events, or the hazard of disease, which concerns the modeling of time until an event of interest, such as a diagnosis or failure.

Case-control studies are analytical epidemiologic studies in which individuals with the health outcome or disease of interest are compared with a suitable control group of individuals without the health outcome or disease to determine differences in exposure levels between the groups. Case-control studies are typically retrospective in design, meaning that they evaluate exposures in the past, with exposure assessment occurring after disease onset in cases.

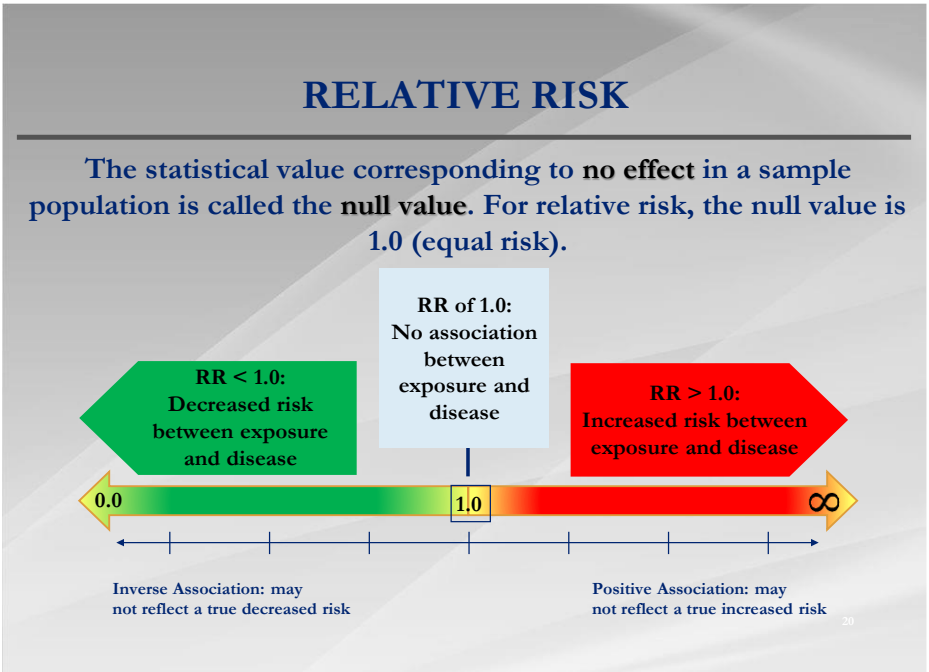
Cross-sectional studies examine the correlation between an exposure and the prevalence of a disease in a defined population at a specified point in time, thereby providing a "snapshot" of the potential exposure-disease relationship at a particular time. Because exposures and outcomes are typically assessed simultaneously in a cross-sectional study, it often is not possible to determine whether the exposure preceded or followed the disease, thereby obscuring cause-and-effect relationships. Thus, cross-sectional surveys are considered lower quality designs compared with the more analytically rigorous cohort and case-control designs and are insufficient to establish causation on their own.

Ecologic studies are epidemiologic studies in which the units of analysis are populations or groups, rather than individuals. Observations at the group level may not apply to individuals; for example, general or average exposures do not apply to every member of a population. Erroneous conclusions can be reached if inferences about individuals are made based on aggregate observations—a logical error known as the ecologic fallacy. An example of the ecologic fallacy would be found in a study evaluating the association between prenatal exposure to influenza and development of childhood acute lymphocytic leukemia (cALL). Data collected from prenatal records and cancer registries in a metropolitan area might reveal a statistically significantly increased risk for cALL with prenatal influenza exposure. However, given that the researchers did not collect individualized exposure data, they were uncertain if the mothers of the cALL patient were exposed to prenatal influenza (Gordis 2009). Another major limitation of ecologic studies is the impact of confounding and bias. Since information is collected at the aggregate, rather than the individual level, the impact of confounding cannot be controlled adequately. However, ecologic studies may be used for generating hypotheses and informing policy decisions that affect entire groups if findings are confirmed by analytical studies. This does not, however, make them a valid basis for evaluating causation.

Estimates of Risk

In general, all the above-mentioned study designs can yield estimates of **relative risk (RR)**, that is, the ratio of the probability of an outcome occurring in an exposed group compared with an unexposed group. RR is a general term that may refer to a risk ratio, a rate ratio, an odds ratio, a prevalence ratio, a standardized incidence or mortality ratio, or a hazard ratio. The type of RR estimate calculated depends on the study design and data available. An RR equal to 1.0 means that the probability of the outcome is equal in the exposed and unexposed groups (referred to as no association or a null association). An RR greater than 1.0 means that the probability of the outcome is greater in the exposed than the unexposed group (referred to as a positive association), whereas an RR less than 1.0 means that the probability of the outcome is lower in the exposed than the unexposed group (referred to as an inverse or negative association) (Elwood 1998, Gordis 2009, Szklo and Nieto 2007).

Interpretation of Relative Risk Estimates



Less commonly, epidemiologic studies estimate a **risk difference**, that is, the absolute difference in the probability of an outcome in an exposed group compared with an unexposed group (e.g., the incidence rate in the exposed minus the incidence rate in the unexposed). A risk difference equal to 0.0 means that the probability of the outcome is equal in the exposed and unexposed groups; a risk difference greater than 0.0 means that the probability of the outcome is greater in the exposed group, whereas a risk difference less than 0.0 means that the probability of the outcome is lower in the exposed group.

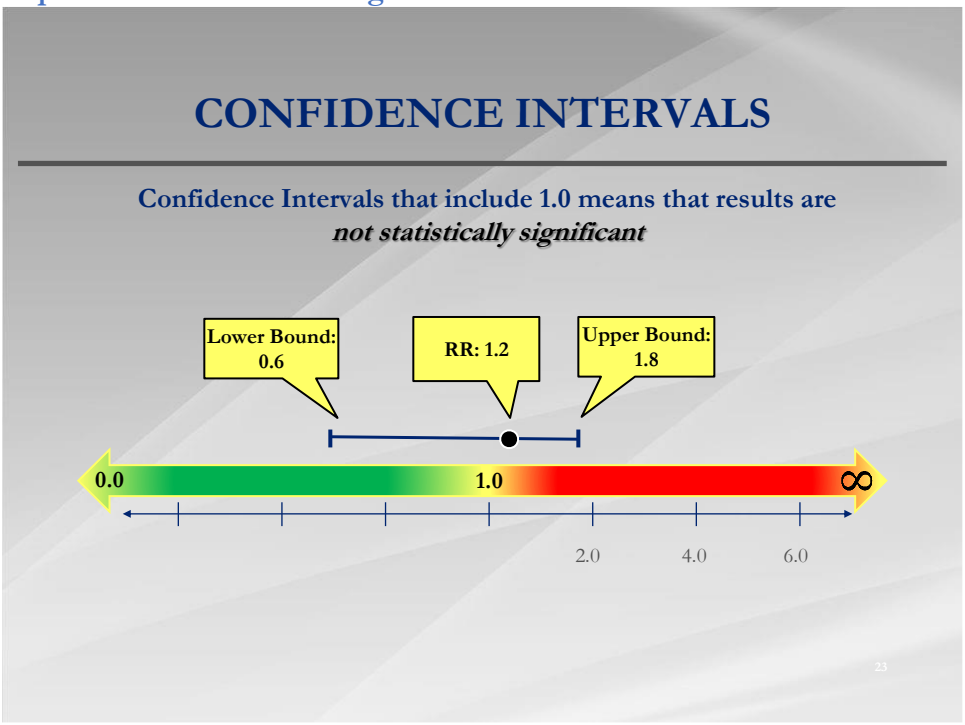
Estimates of relative and absolute risk, as well as incidence rates, mortality rates, and prevalence, should be reported with a margin of error, which in epidemiology and statistics conventionally takes the form of a confidence interval. In a valid and unbiased analysis, a confidence interval will, over repeated sampling of the study population, contain the value of interest with a frequency no less than its stated confidence level, which in epidemiology and statistics is typically set at 95%. The width of a confidence interval provides an indication of the precision of an estimate. Thus, a relatively wide confidence interval denotes a relatively inexact or statistically unstable estimate, whereas a relatively narrow confidence interval denotes a relatively precise estimate (Rothman, Greenland, and Lash 2008).

The **statistical significance** of an observed finding may be denoted by a P-value or a confidence interval. A **P-value** is the probability of observing the result or a more extreme result if the null hypothesis of no exposure-disease association were true (Federal Judicial Center 2011). In general, it denotes the probability that the observed condition (or a more extreme one) could have occurred by chance alone. In epidemiology, a standard P-value cut-off for statistical significance is 5%; that is, a result whose P-value is less than 5% is considered sufficiently unlikely to have occurred by chance

and that it is designated as "statistically significant." Likewise, if a 95% confidence interval excludes the null value of the estimate (e.g., 1.0 for an RR or 0.0 for a risk difference), then the result is said to be statistically significant, with less than a 5% probability that the result occurred by chance.

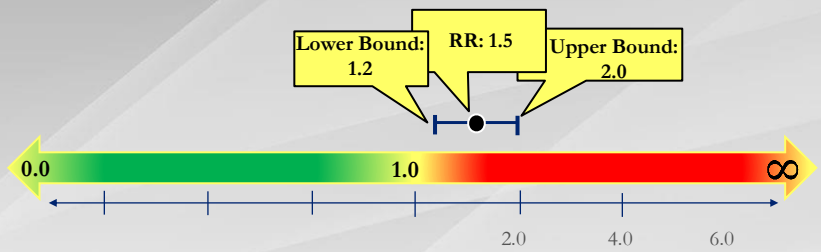
In epidemiology RRs are rarely 1.0. Thus, statistical significance testing is necessary to determine whether the association may have occurred by chance. In addition, other factors, such as confounding and bias, may produce a flawed estimate of relative risk. These concepts are discussed below.

General Interpretation of Statistical Significance based on Confidence Intervals



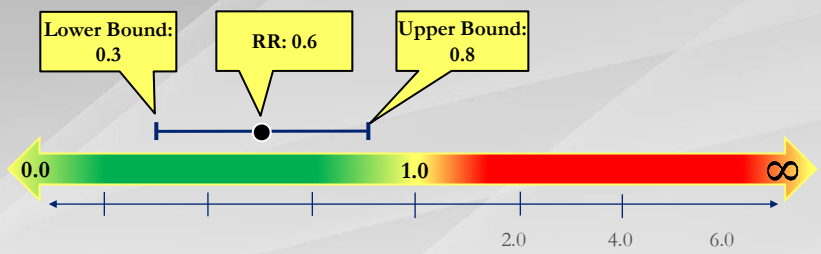
CONFIDENCE INTERVALS

Confidence Intervals with both bounds above 1.0 means that results are ***statistically significantly positive***



CONFIDENCE INTERVALS

Confidence Intervals with both bounds below 1.0 means that results are ***statistically significantly inverse***



Association versus Causation

Properly conducted analytical epidemiologic studies can identify statistical associations between an exposure, such as a chemical exposure, and a disease or other health outcomes. However, this is merely the first step in the causal analysis; a statistical association does not by itself indicate a causal relationship, that is, that the exposure caused the disease or health outcome. Whether a given association is causal should first be evaluated in light of possible alternative explanations including bias, confounding, and chance. Then, once those are evaluated, other criteria are applied—such as the Bradford Hill criteria that I discuss below—to make a scientifically valid determination of causation (Rothman, Greenland, and Lash 2008, Baker and Nieuwenhuijsen 2008, Elwood 1998, Gordis 2009, Szklo and Nieto 2007).

Bias refers to any systematic or random error in the design, conduct, analysis, or production of a study that can invalidate the study's results and interpretation. One common type of bias is **selection bias**, which refers to systematic differences in characteristics, specifically those that are associated with exposure and disease status, between people who participate in a study and those who do not. For example, selection bias can occur in a case-control study if the participation rates differ between cases and controls for reasons that are related to the exposure of interest. Selection bias can occur in a cohort study if exposed and unexposed persons drop out of the study at different rates for reasons that are related to the disease of interest. For example, selection bias could occur in a cohort study on air pollution if individuals in the exposed area migrate away from the area after developing symptoms (Baker and Nieuwenhuijsen 2008). Another example is residential migration from outside of the exposure area of interest prior to disease diagnosis to an exposed area where a diagnosis is made shortly thereafter. In this scenario, exposure would not be related to disease occurrence because the relevant exposure window occurred outside of the study area.

Information bias, also referred to as misclassification bias, refers to systematic errors in data collection that result in unequal accuracy of information, such as exposure and disease status, between comparison groups. Types of information bias include recall bias (i.e., discrepancies in the accuracy or completeness of recollection of past events or experiences between comparison groups), interviewer bias (i.e., differences in data collection or recording methods between comparison groups based on an interviewer's knowledge of the subjects' exposure or disease status), and response bias (i.e., differences in the distribution of missing data between study groups, such as whether or not a participant responds to survey questions). For example, an individual living in close proximity to a factory may report more respiratory symptoms than an individual living a great distance from the factory, simply because the individual in close proximity to the factory is more concerned about air pollution. Another type of information bias is detection bias or surveillance bias, for which individuals who are aware that they are under observation may be more likely to report symptoms or outcomes, or seek medical care leading to a diagnosis or detection of a condition.

Confounding refers to a distortion of the estimated association between an exposure and a disease by a third, extraneous factor, called a confounder. A **confounder** is an independent risk factor for the disease that is unequally distributed between exposed and unexposed individuals and is not affected by the exposure. Failure to control for confounding can invalidate the estimated exposure-outcome association. In most observational epidemiologic studies, investigators attempt to control

for confounding by using a statistical procedure called adjustment. To adjust statistically for confounding, it is necessary for a study to have collected sufficient information on potential confounders. Even if a study has adjusted for a confounder, residual confounding can occur if insufficient information about the confounder has been collected or if the confounder is inappropriately represented in a statistical model. In studies of certain diseases for which little is known about causes, there may be unknown risk factors that confound the exposure-outcome relationship under study.

In epidemiology, **chance** refers to random, unpredictable error due to sampling variability or imprecise measurement of study variables, resulting in fluctuation around a true value. This can occur when the time period and geographic delineation of a study (or case observation period in time and space) has not been clearly determined. Chance can never be completely excluded as a potential explanation for an observed finding, although the probability of a chance finding can be reduced by increasing the sample size, improving the precision of a measurement tool, or increasing the number of measurements. Importantly, these are all components of analytical methodology that involves case ascertainment, denominator enumeration, comparison populations, evaluating of bias and confounding, and statistical testing. Chance findings may also occur from the random variation of data when multiple statistical tests are being conducted, such as in genetic polymorphism studies or drug trials. Of note, it would be expected that random chance findings would occur in studies investigating all PFAS, given that upwards of thousands of specific PFAS types exist.

As noted above, establishing whether a given association is causal requires more than a mere statistical association or relative risk estimate, even when the foregoing factors of bias, confounding, and chance are controlled. It also requires an evaluation of the overall weight and consistency of the relevant epidemiologic evidence, which may be combined with sufficient supporting evidence from toxicology and an understanding of underlying plausible and established biological mechanisms by which the exposure may be causing disease. In order to take the next step and provide a framework for the complex process of determining whether a particular association is causal, epidemiologists have developed guidelines to assist them in making this assessment. Sir Austin Bradford Hill, a pioneering epidemiologist and statistician in the mid-20th century, developed guidelines to help determine whether a statistically significant association was causal: "Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?" (Hill 1965). Hill specified nine guidelines for evaluating the current state of knowledge regarding an exposure-disease association: strength of association, consistency, specificity, temporality, biological gradient, biological plausibility, coherence, experiment, and analogy (Hill 1965).

1. Strength of association refers to the magnitude of the estimated effect of the exposure on the disease. Although weak causal associations and strong non-causal associations certainly exist, a strong association between an exposure and a disease outcome is less likely than a weak association to be explained by confounding or other sources of moderate bias.

2. Consistency refers to repeated observations of an exposure-disease association in various populations, a pattern that can lend credibility to a causal interpretation, although consistency across settings may not be observed if a causal effect occurs only under unique circumstances.

3. Specificity refers to the restriction of an association to a single exposure with a single effect, which may support a causal interpretation.

4. Temporality refers to the fact that in any causal relationship, the cause must precede the effect in time. (This is the only one of Hill's nine guidelines that is essential to establishing causality.)

5. Biological gradient refers to a monotonic relationship (i.e., a gradient that does not change direction) between an exposure and a response, which can support a causal interpretation, although it is neither necessary nor sufficient to establish causality.

6. Biological plausibility of an exposure-disease relationship depends on current knowledge in toxicology, biology, and other fields. Such knowledge is constantly evolving, and what is considered a plausible causal hypothesis is somewhat subjective.

7. Coherence refers to compatibility of a causal hypothesis with what is generally known about the natural history and biology of a disease. However, such information may be unavailable, incorrect, or misinterpreted.

8. Experiment refers to the notion that it is occasionally possible to use experimental or semi-experimental epidemiologic evidence, such as intervention studies, to support a causal association; however, such evidence is often lacking or not relevant.

9. Analogy refers to the consideration of similar exposure-disease relationships, but analogies cannot be used to prove or disprove a causal hypothesis.

Hill referred to these as nine "features to be specially considered" or "viewpoints" on the basis of which one should evaluate associations before declaring them causal. He did not assert that all guidelines must be met to establish causality; rather he stated: "None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non" (Hill 1965) ("Temporality" is the exception, as noted above.) Although other approaches for evaluating causality have been described, the Hill guidelines (in various revised forms) are commonly cited and implemented by epidemiologists (Hennekens and Buring 1987, Gordis 2009, Mausner and Kramer 1985, Rothman, Greenland, and Lash 2008, Lilienfeld and Stolley 1994, Schlesselman 1982) and are broadly accepted in the scientific community. The Hill guidelines are also widely relied upon by courts for assessing general causation, that is, for determining whether the weight of scientific evidence shows that an exposure is capable of causing a disease and, if so, the circumstances under which such causation occurs (Cole 1997, Green, Freedman, and Gordis 2011).

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December 14, 2020

RE: *Kevin D. Hardwick, et al. v. 3M Company, et al.*

Appendix B: General Description of Study Population Sources

The following sub-sections provide a general overview of the some of the sources of study populations for which PFASs have been evaluated. These summaries are not intended to provide an exhaustive list of all studies conducted among the study population sources. In addition, these summaries do not discuss the analytical results or findings within and across populations.

Occupational Studies

3M Workers

The majority of studies of 3M workers have been published evaluating study populations from production facilities in the Minneapolis/St. Paul, MN area, and in the Decatur, AL area. The primary exposure of interest from the MN facilities is PFOA while the primary exposure of interest in the AL facilities is PFOS. However, exposure to both PFOS and PFOA compounds may occur at multiple facilities.

Gilliland (1993) (Gilliland and Mandel 1993) conducted a retrospective cohort mortality study of 2,788 male and 749 female workers at a 3M PFOA production plant in Cottage Grove, MN. The facility included several divisions, and PFOA production (in addition to the use of other chemicals) was limited to the Chemical Division. To be eligible for study inclusion, workers were required to be employed at the plant for at least six months for any period between January 1, 1947 and December 31, 1983. Vital status was ascertained from the Social Security Administration and from the National Death Index. The cohort was followed through 1989. Mortality was confirmed using death certificates obtained from State Health Departments and information on two deaths that occurred outside of the United States was obtained via family members. A nosologist coded the underlying causes of death according to the ICD, 8th revision. To be classified as exposed, an individual was required to have worked in the Chemical Division for at least one month. Unexposed workers were those who did not work in the Chemical Division or who worked in this division for less than one month. Year and age at first employment, duration of employment, and months in the Chemical Division were used as surrogate measures of cumulative exposure. The observed number of deaths was compared with sex, race, age, and calendar period adjusted cause-specific mortality rates of the U.S. and Minnesota populations. Vital status was obtained for 100% of the analytical cohort.

In a follow-up to Gilliland (1993), Lundin (2009) (Lundin et al. 2009) extended the enrollment period, analytical duration, and conducted more internal analyses based on estimated exposure-response categorizations. Specifically, the period of enrollment was extended from 1983 to 1997 and analytical follow-up was extended from 1989 through 2002. In the former study, eligibility for entry into the cohort was based on employment for at least six months; however, Lundin (2009) required a minimum of 365 days of employment prior to the end of 1997. The authors suggested that this

methodological decision was made to exclude the "relatively large number of short-term workers, many of whom were summer interns." Employment data for an additional 169 employees eligible for both studies were also identified. A total of 807 deaths were identified in the current study, while 398 decedents were identified in the former. Vital record searches were performed through the National Death Index, and the underlying cause of death was coded according to the ICD revision in effect at the time of death. The likelihood of exposure for each job held was based on review of work history records and expert historical knowledge of the manufacturing process. An expert panel of industrial hygienists and veteran workers reviewed job and process information, and ultimately, three categories of exposure were generated as follows:

• Definite occupational exposure: "Primarily jobs where electrochemical fluorination, drying, shipping, packaging, and quality-control analyses of ammonium perfluorooctanoate occurred. Workers were exposed on a regular basis with potential for high exposure."

• Probable occupational exposure: "Jobs in other chemical division areas where ammonium perfluorooctanoate exposure was possible, but likely lower or transient."

• No or minimal occupational exposure: "Jobs primarily in the nonchemical division of the plant. Opportunity for some exposure (more than the general population) due to contamination at the work site."

Two types of analyses by job classification were conducted. The first compared the mortality of workers in the above job categories with the expected rates based on the general population of MN. The second analysis was based on internal comparisons evaluating workers in high or moderate exposure jobs for six months or more, compared with low exposure workers. In addition, the authors conducted internal comparison analyses based on weighting factors for exposure intensity in certain job classifications. These estimates were based, in part, on serum concentrations collected in 2000 from a sample of workers. Cumulative exposure for each worker was calculated as a sum of the days of employment at each exposure level multiplied by the exposure weighting factor. The internal analyses adjusted for sex, year of birth, age at entry into the cohort, smoking status, and wage type. The exposure models were lagged by 10 years. More workers with definite exposure (compared with non-exposed) were smokers, although complete smoking data for both groups were lacking. Most workers with definite exposure jobs were hourly employees, while most non-exposed workers were salaried employees.

Raleigh (2014) conducted an updated mortality analysis and a new cancer incidence analysis of the Cottage Grove, MN cohort and a non-exposed comparison population from a 3M company plant in St. Paul, MN (Raleigh et al. 2014). Similar to the 2009 study, employees eligible for study inclusion were required to work a minimum of one year at the facility. A total of 4,668 eligible employees were included from the Cottage Grove plant, including 675 hired between 1997 and 2002. A referent cohort of 4,359 workers from a non-PFOA exposed 3M production facility in St. Paul was assembled. These two study populations were similar in that they were employed by the same company, located in the same geographic region, and were represented by the same labor union. Mortality records of all workers were available through 2008. Incident cancer cases were identified through linkage with the Minnesota Cancer Surveillance System and the Wisconsin Cancer Reporting System. Exposures were estimated via work history records, industrial hygiene monitoring data, industrial hygiene professional, worker interviews, and average annual APFO production levels. Time-dependent exposure was modelled using Cox regression analyses.

Alexander and colleagues conducted a retrospective mortality cohort study in 2003 of 2,083 workers who had at least one year of cumulative employment from 1961 to 1997 at a manufacturing facility in Decatur, Alabama, and a follow-up in 2007 (Alexander et al. 2003, Alexander and Olsen 2007). The facility consisted of two plants: a film plant and a chemical plant with workers possibly being exposed to perfluorooctanesulphonyl fluoride (POSF). Although POSF-based chemicals were the major fluorochemical manufactured, exposure to other fluorochemicals, including PFOA, were also likely. Workers were categorized into 3 exposure categories: high, low or no potential workplace exposures to POSF-based fluorochemicals based off the serum samples of 186 (126 chemical plant, 60 plant) workers and worker job categories. The geometric mean serum PFOS level for chemical plant employees was 0.9 ppm (900 ppb), and the geometric mean serum PFOS level for film plant employees was 0.1 ppm. Age, gender, and calendar period adjusted mortality rates were calculated for workers from the date they had one year of employment through 1998 using two reference populations: the state of Alabama and 23 regional counties. Further exposure specific analyses were conducted among workers who had at least one year of cumulative employment in job with high or low exposures to POSF-based fluorochemicals. Olsen (2004) examined health claims data for information about episodes of care from 1993 through 1998 among two groups in the Decatur, Alabama worker cohort: 652 chemical plant workers and 659 film plant workers (Olsen et al. 2004). An episode of care was defined as a health problem that was related to a series of related events with a start and finish and was categorized based on ICD diagnosis codes, current procedural terminology codes from facility and professional health claims, revenue, and National Drug codes. Serum PFOS concentration levels among workers were determined through the use of a job-exposure matrix similar to the one used in Alexander 2003. Workers were categorized into 1 of 3 groups based off their work history: working in the chemical plant (n = 652), the film plant (n = 659) or both from 1993 to 1998. Each group was indirectly standardized for age (<40, 40-49, ≥50) and sex and the number of health claims were compared to the number of claims expected from the remainder of the 3M United States manufacturing population. A ratio of the two indirect standardized estimates was calculated (risk ratio episodes of care (RRE_pC)). A separate similar analysis was conducted among long-term workers that compared those who worked in high-exposure chemical plant jobs (n = 211) to those who worked in non-POSF exposed similar jobs in the film plant (n = 345) from 1983-1993.

DuPont Workers

Leonard (2008) (Leonard et al. 2008) conducted a retrospective cohort study among the DuPont Washington Works (WW) employee cohort. This study followed up on a 2004 cross-sectional health survey of 1,025 active employees at the WW plant. This 2004 study found that all participants in both processing and administrative areas had detectable levels of serum PFOA. The WW cohort consists of individuals who worked at the DuPont WW polymer production facility in Parkersburg, West Virginia between its opening on January 1, 1948 and December 31, 2002. Unlike the DuPont Epidemiology Registry, which monitored rates for only current DuPont workers, all workers who had worked at the WW plant during the period of eligibility were included in the WW cohort. A total of 6,027 individuals with a history of work at the plant were included in this cohort; 21 of whom transferred to the WW location after December 21, 2002, and one of whom had an unverifiable birth date and had to be excluded from analysis. A total of 5,476 of the workers at the WW plant were already registered in the DuPont Epidemiology Registry, and an additional 573 individuals were identified using work history records from the WW plant. Social Security numbers

were used to confirm vital status of cohort members as of December 2002. The mortality rate within the WW cohort was compared to the mortality rates of subjects in 5-year age categories using three external reference populations: the United States, West Virginia, and other DuPont workers from Indiana, Kentucky, North Carolina, Ohio, Pennsylvania, Tennessee, Virginia, and West Virginia, excluding those in the WW cohort. Expected mortality rates were calculated based on population distributions by sex, age, and time periods for subjects in 5-year age categories between 1948 and 2002. Standardized mortality ratios (SMRs) were calculated for all causes of death, all malignant neoplasms, individual cancers, infectious diseases, diabetes, cerebrovascular disease, heart disease, individual types of heart disease, non-malignant respiratory disease, cirrhosis, kidney disease, and external causes of death including accidents, homicides, and suicides.

Sakr (2009) conducted a more specific retrospective cohort study on this workforce by focusing on risk of ischemic heart disease (IHD) (Sakr et al. 2009). This analysis included 4,747 workers. Detailed work history information for all employees was used to estimate the time dependent APFO exposures using an exposure reconstruction model developed through the DuPont Epidemiology Program. Serum biomonitoring was used as an indicator of exposure. A job exposure matrix was validated using historical blood data collected from 1979 to 2002 from voluntary participants in a different biomonitoring program at the WW plant, which collected samples to monitor workplace exposure and ensure effective exposure controls. Each worker's cumulative exposure was calculated by multiplying the duration of time in each work assignment by the mean intensity estimate from the job exposure matrix and taking the sum of all period exposure estimates. Cox proportional hazards regression was used to evaluate time dependent exposures.

Steenland (2012) conducted an updated mortality analysis of 5,791 workers in the DuPont Washington Works employee cohort with follow-up extending to 2008 (Steenland and Woskie 2012). A job-exposure matrix was created by separating workers into 5 job category groups based on indirect or direct PFOA exposure and area of work: 1) direct PFOA exposure in the Teflon production area, 2) direct PFOA exposure in the other copolymer production areas (fluorinated ethylene propylene and perfluoroalkoxy fluoropolymer operations), 3) intermittent direct non-PFOA use jobs, 4) maintenance jobs with intermittent direct or plant background PFOA exposures, or 5) non-Teflon/copolymer production division jobs with no PFOA use. Measured serum PFOA levels of 1,308 workers with at least one year of experience in the above job categories at the time of sampling were collected from 1979 to 2004. The estimated average serum PFOA level was 350 ng/mL. The mortality rates were calculated using two external reference populations: mortality rates from other DuPont workers in the Appalachian region from 1955-2009, and mortality rates from the United States national population from 1940-2007 with extrapolation to 2009. Exposure-response analyses used cumulative serum levels (ppm-years) and quartiles were created from the cumulative serum levels of decedents. Separate analyses were conducted by applying different latency periods (0, 10 and 20 years). Steenland (2015) conducted a retrospective cohort study of a subset of workers who were part of the 2012 DuPont cohort mortality study and had retrospective exposure estimates (Steenland, Zhao, and Winquist 2015). Workers (3,713) were interviewed from 2008 to 2011 (19.3% being interviewed once, while the remaining were interviewed twice) to collect demographic, smoking, residential, medical and reproductive histories. Information on incident diseases was collected through medical record validation and three self-reported disease outcomes. A similar job-exposure matrix to the one in Steenland 2012 was used, with it being based on over 2000 serum samples collected from 1979-2004 from workers who had at least one year of experience in the job category at the time of sampling. Retrospective serum estimates for community residents from PFOA-contaminated water were obtained through a pharmacokinetic model that used

information generated from an environmental fate and transport model about yearly PFOA estimates from air, surface and ground water, information about residential histories from the 2005/2006 C8 Health Project, and information from surveys, public water supply network maps and drinking water sources. The median measured serum level was 113 ng/ mL in 2005.

Other Occupational Study Populations

Consonni (2013) conducted a Multicenter Mortality Study of Tetrafluoroethylene (TFE) production workers in 4 companies with 7 production sites, including the DuPont West Virginia facility (Consonni et al. 2013). Tetrafluoroethylene is a compound used in the production of fluorinated polymers, including polytetrafluoroethylene (PTFE). This process uses ammonium perfluoro-octanoate (APFO), the ammonium salt of PFOA. APFO readily forms PFOA in aqueous solutions. The authors examined the cause-specific mortality rates in this worker cohort between 1950 and 2008. The cohort consisted of 5,879 male workers– 4,773 ever exposed, and 1,081 never exposed. Some workers from the aforementioned DuPont cohorts were included in the analyses. A job-exposure-matrix was ultimately developed with TFE and APFO measures for the relevant job titles at each site from 1950-2002. Vital status ascertainment and underlying cause of death was performed by record linkage or individual follow-up. For each worker, person-years at risk were calculated from the time they satisfied both entry requirements – minimum employment and acquiring the relevant exposure. Follow-up ended at date of death, loss to follow up, or study end, whichever came first. Individual occupational histories were merged with the plant-specific JEM to calculate time-varying cumulative exposures. Overall SMR analyses and cumulative exposure analyses were performed for workers exposed to APFO.

In 2009, Costa conducted a study of health outcomes of workers in a PFOA production facility in Trissino, Italy from 1978-2007 (Costa, Sartori, and Consonni 2009). Fifty-three male workers from the PFOA production department were included, and 12 executive clerks and 95 blue collar workers served as unexposed controls. PFOA workers were categorized into currently exposed (n = 37) or previously exposed (n = 16). All subjects underwent yearly physical examinations, including blood chemistry tests for hematology, liver, renal functions, glucose and lipid metabolism, and uric acid. In 2002 and 2006 only, PFOA workers were also tested for Apo-A, Apo-B, lipoproteins, C-reactive protein, immunoglobulins, sex hormones, and thyroid function. Biological monitoring of serum PFOA started in 2000, and was repeated yearly with the exception of 2005 for both current and former PFOA workers. A matched pair analysis of all serum levels was done, matching age, work seniority, day/shiftwork, and living conditions. Regression models testing the relationship between PFOA exposure and health outcomes adjusted for covariates, including age, years of exposure, year of PFOA sampling, BMI, smoking and alcohol consumption. All workers were exposed to very high levels of PFOA (see forthcoming bar chart).

Community Studies

Vieira (2013) evaluated the association between cancer risk and PFOA exposure in Ohio and West Virginia residents living near the DuPont Teflon manufacturing plant in Parkersburg, West Virginia (Vieira et al. 2013). PFOA was released into the environment beginning in the 1950s, contaminating both private and public water sources. The study population consisted of 25,107 incident cancer cases diagnosed between 1995 and 2005 in five Ohio counties and eight West Virginia counties identified from state registries and geocoded down to the street or zip code level. Control subjects

for each cancer were registry-based cases of all other cancers except that cases of kidney, pancreatic, testicular, and liver cancer were excluded as controls. Odds ratios adjusted for age, race, sex, diagnosis year, insurance provider, and smoking status were calculated for 18 cancers and association with living in an exposed water district as well as by individual-level estimated annual PFOA serum concentration. Modeled PFOA serum concentrations by water district ranged from 5.3 to 125 ug/L while estimated individual-level annual PFOA serum concentrations ranged from 3.7 to 655 ug/L, assuming a 10-year residency and 10 years latency.

Barry (2013) evaluated risk of cancer in 28,541 Ohio residents from the C8 Health Project exposed to PFOA in drinking water and 3,713 DuPont workers, approximately half of whom had both residential and occupational exposure to PFOA (Barry, Winquist, and Steenland 2013). For community residents, the median measured serum PFOA level in 2005-2006 was 24.2 ng/mL (range: 0.25-4,752 ng/mL) and median estimated annual PFOA serum level was 19.4 ng/mL (range: 2.8-9,217 ng/mL). The corresponding median measured and estimated PFOA serum levels for DuPont workers were 112.7 ng/mL (range: 0.25-22,412) and 174.4 ng/mL (range: 5.2-3,683), respectively. Only validated primary cancer cases were included in the analysis. Hazard ratios were adjusted for time-varying smoking, time-varying alcohol consumption, sex, education, and stratified by 5-year birth periods. Hazard ratios were calculated for each cancer per unit of log estimated cumulative PFOA serum concentration. Hazard ratios for cancer of the kidney, testes, and thyroid were also evaluated by estimated quartiles of serum PFOA concentration.

Numerous studies of PFAS exposure (primarily PFOA and/or PFOS) have been conducted among general population study samples, such as those based on data collected from the National Health and Nutrition Examination Survey (NHANES). NHANES is collection of databases representing the health and nutritional status of adults and children in the U.S. Data are ascertained via interviews and physical examinations. These studies are typically intended to be representative of the general population, and as such, are considered to reflect background levels of PFAS exposures. As a result, they are of relatively limited capacity in evaluating potential dose-response relationships between measured PFOA and/or PFOS exposures and health conditions and clinical markers because the exposure ranges are narrow. Furthermore, many of these studies utilized cross-sectional study designs and as such, provide no basis to evaluate potential temporal relationships.

Targeted Study Populations

Cohorts of select populations have been reported in the scientific literature regarding their PFAS/PFOA/PFOS exposure levels and potential associations or correlations with various health conditions. For example, these populations include fishermen (Bloom et al. 2010), Inuit communities in Greenland and Quebec, pregnant women, residents of specific communities, and cohorts of children. Collectively, these studies have typically evaluated background or near background levels of PFOA/PFOS exposure. Selection bias (generalizability of study findings) is a foremost limitation in many of these studies.

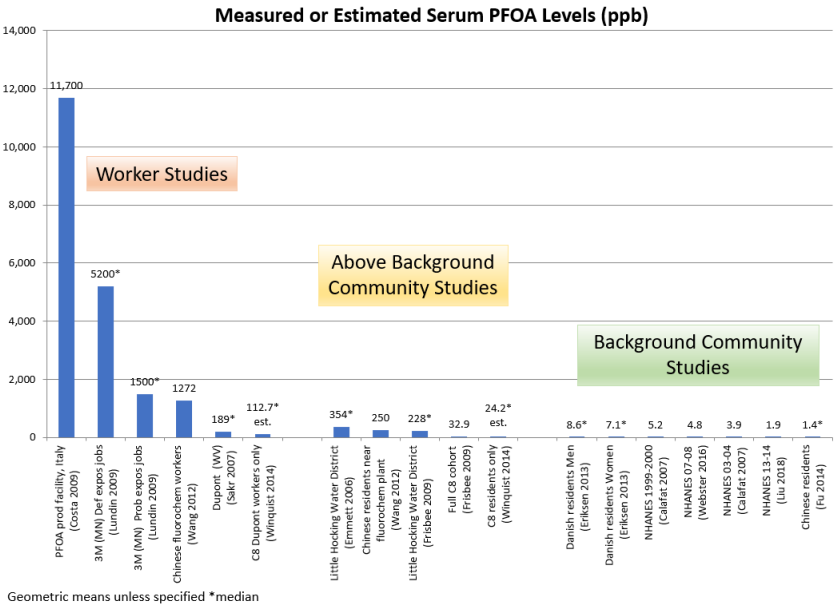
General Exposure Estimates Across Study Population Sources

Based on exposure estimates across the various study population sources, workers in PFOA or PFOS occupational settings have had considerably higher levels of PFOA/PFOS exposure compared to residents in above-background exposure communities and compared to studies that are generally representative of background exposure levels (e.g., NHANES). Interpretation of findings within studies and between study populations should account for the often extensive variation in

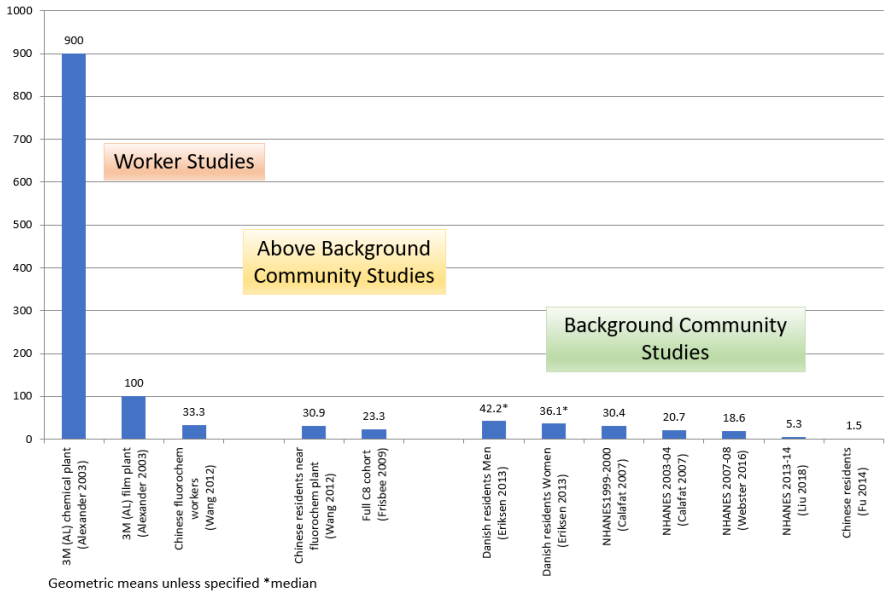
exposure levels. Thus, if no association is observed for a health outcome among workers with the highest PFOA or PFOS exposure, then there would be no valid scientific basis to formulate a conclusion that populations with lower (or near background) levels of exposure would be at increased risk of the outcome. However, if studies show that workers with the highest exposure levels are at increased risk of a specific health outcome, the interpretation should be made in the context of patterns of relative risk in other (similarly exposed) studies and within the context of conclusions drawn for the study population of interest. In other words, evidence of risk may only be found at high exposure levels and the exposures across community settings may be not be associated with an increased risk of disease, even though some positive associations are reported.

Of important scientific relevance, Steenland (2015) states that regarding PFOA exposure: "High-exposure groups are often the most useful for studying human health effects. Workers often have higher PFOA exposures than the general population. A subset of the 3713 workers considered here (n = 1881), who had their serum PFOA measured in 2005/2006, had a median serum level of 113 ng/mL, much higher than the surrounding community (median 24 ng/mL), and the general US population (4 ng/mL)" (Steenland, Zhao, and Winquist 2015).

The following two graphs *generally* represent the measured or estimated serum PFOA and PFOS levels across the various types of study populations. Clearly, exposure levels among the worker populations are considerably higher than other study populations, particularly community studies with background or near-background levels of exposure.



Measured or Estimated Serum PFOS Levels (ppb)



Geometric means unless specified *median

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December 14, 2020

RE: *Kevin D. Hardwick, et al. v. 3M Company, et al.*

Appendix C: State-of-the-Epidemiologic Science of Human Disease, Health Conditions, and Adverse Clinical Markers among PFAS-exposed Study Populations

I have performed systematic reviews of the epidemiologic literature on PFAS exposure and risk of human disease and adverse health conditions. For the purpose of this report, I am not summarizing the hundreds of epidemiologic studies on PFAS exposure; rather, I provide a brief overview of my process for review and the general and overarching conclusions and methodological considerations concerning PFAS exposure and risk of human disease, health conditions, and adverse clinical markers. I do, however, provide a comprehensive reference list representing the totality of epidemiologic studies on PFAS exposure and human health outcomes.

My systematic review of the literature was conducted with respect to well-established guidelines for the conduct of comprehensive evaluations of epidemiologic studies. Specifically, the identification of studies, inclusion and exclusion criteria, critical examination of internal (e.g., information bias, confounding) and external (e.g., selection bias) validity, synthesis and analysis of study results data, and the interpretation of the collective body of epidemiologic evidence was conducted according to principles and protocols delineated in Meta-analysis Of Observational Studies in Epidemiology (MOOSE) (Stroup et al. 2000), Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Moher et al. 2009), and Strengthening the Reporting of Observational studies in Epidemiology (STROBE) (von Elm et al. 2007). While acknowledging that a clear association should exist prior to performing a causal assessment (Hill 1965), my *a priori* methodology included the consideration of key factors for making claims of general causation, such as the strength of association, consistency of findings across studies, and potential dose-response patterns.

I performed comprehensive electronic literature searches using the National Library of Medicine PubMed database to identify potentially relevant articles that provided risk estimate data for cancer and non-cancer disease outcomes among PFAS-exposed study populations. It is noteworthy that the large majority of analytical epidemiologic studies of PFAS-exposed populations focused on PFOA and/or PFOS exposure. Thus, the overwhelming majority of data across the analytical epidemiologic studies, including the occupational and above-background community studies, pertain to PFOA and/or PFOS. Literature searches were also conducted to identify articles that reported measures of associations or correlational data (e.g., linear regression coefficients) for clinical markers among PFAS-exposed study populations. Over 2,000 articles pertaining to PFAS exposure and health outcomes were identified in the first phase of the electronic literature search.

All included studies were carefully reviewed, focusing on the qualitative information and quantitative data according to: author and year of study, geographic study area, nature of the cohort, study size, years of follow-up, PFAS/PFOA/PFOS exposure definition, method of exposure assessment, exposure metric units, analytical comparison of exposure metrics, reference populations, number of exposed cases, relative risk estimates, 95% CIs, and the variables that were statistically adjusted for,

if any. A thorough examination of methodological information regarding the potential impact of bias and/or confounding on the interpretation of each study was conducted.

General and Overarching Opinions on PFAS Exposure and Risk of Human Disease

I have conducted a state-of-the-epidemiologic science review of per- and polyfluoroalkyl substances (PFASs), focusing on, but not limited to, perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS), which are the two most frequently studied compounds across the epidemiologic literature, and risk of human disease, health conditions, and adverse clinical markers. I followed well-established systematic methodology to identify, analyze, and interpret the available epidemiologic evidence regarding the diseases, health conditions, and clinical factors of interest.

Disease Outcomes

Based on the totality of epidemiologic data, the evidence does not support an independent association between PFASs exposure, including PFOA or PFOS exposure, and increased risk of any human disease or health condition. Thus, the epidemiology does not support a conclusion that exposure to any PFAS, or PFOA or PFOS exposure specifically, is a general cause of any disease or health condition. In particular, the patterns of associations across studies do not support a conclusion that PFAS exposures independently increase the risk of any disease or health condition.

a. *Cancer:* The analytical epidemiologic evidence is not supportive of an increased risk of any type of cancer among PFAS-exposed study populations, including testicular cancer, kidney cancer, bladder cancer, breast cancer, liver cancer, prostate cancer, thyroid cancer, pancreatic cancer, non-Hodgkin lymphoma, multiple myeloma, or leukemia. This conclusion is based on a critical and systematic evaluation of internal validity parameters, patterns of relative risk estimates, consistency of findings, and dose-response evaluations. The majority of relevant epidemiologic evidence originates from well-conducted analytical occupational cohort studies among workers with high levels of PFOA and PFOS exposure.

b. *Cholesterol and Cardiovascular Outcomes:* Although findings are somewhat inconsistent, several studies have reported elevations in total cholesterol and LDL among PFOA and PFOS exposed study populations. However, most studies show no relationship between PFOA or PFOS exposure and HDL levels. These results are inconclusive given that many studies utilized a cross-sectional design that does not account for changes in PFOA/PFOS exposure, changes in variables that impact cholesterol levels (e.g., dietary habits over time), and changes in cholesterol levels over time. Furthermore, numerous modifiable and non-modifiable factors impact cholesterol and lipoprotein levels in the human body, such as diet, physical activity, body weight, and genetics. The studies generally do not adequately account for these relevant confounders. In addition, a relatively large proportion of the U.S. population is on cholesterol lowering medication, but many studies do not account for this important factor either. Perhaps most important, these inconsistently observed associations cannot be interpreted as causal given that the analytical epidemiology fails to show an association between exposure to any PFAS and cardiovascular or cerebrovascular disease, including coronary artery disease, stroke, or hypertension. Given the high

correlation between elevated cholesterol levels and heart disease, a lack of correlation between PFAS and these outcomes strongly suggests that any observed correlations with cholesterol are not true causal relationships.

c. *Other Diseases and Health Conditions:* The analytical epidemiologic evidence does not support an independent association between exposure to any PFAS, including PFOA or PFOS exposure, and other diseases or health conditions.

    i. Thyroid disease: While some studies show statistically significant changes in thyroid hormone levels associated with serum PFOA/PFOS exposure, reported thyroid hormone values have generally been within normal reference ranges and are not clinically significant. Furthermore, results from studies of the highest exposed populations (occupational cohorts) are not supportive of an increased risk between PFOA or PFOS exposure and risk of adverse thyroid function. Many lifestyle, dietary, clinical, and environmental factors affect thyroid hormone levels including stress, inflammation, infection, radiation, certain medications (e.g., amiodarone, phenobarbital, lithium, interferon), goitrogenic foods (e.g., cruciferous vegetables, soybeans, cassava root), fluoride, pesticides, mercury, cadmium, lead, pregnancy, liver and kidney dysfunction, and Celiac disease. Collectively, the epidemiologic studies do not adequately control for the confounding influence of all of these factors. The overall weight of the epidemiologic evidence does not support an independent association between exposure to any PFAS, including PFOA or PFOS exposure, and thyroid disease.
    ii. Diabetes: Results across the epidemiologic studies of PFAS exposure, including PFOA and PFOS exposure, and risk of diabetes are inconsistent. While some studies report positive associations for type 2 diabetes, others report no evidence of an increased risk. Furthermore, interpretation of data from some of these studies is limited by the lack of information pertaining to potential temporal relationships and incomplete adjustment for relevant confounding factors. Collectively, the evidence does not support a conclusion that exposure to any PFAS is associated with risk of diabetes, namely type 2 diabetes.
    iii. Osteoarthritis: The epidemiologic evidence does not support an increased risk of osteoarthritis based on exposure to any PFAS. Although interpretation is based on data from few studies, inverse associations for osteoarthritis were observed in the most methodologically and analytical rigorous study, which was conducted among workers with high PFOA exposure.
    iv. Asthma: Several studies of asthma have been conducted on populations exposed to PFASs, including PFOA and PFOS, with some cross-sectional studies evaluating background or near-background levels of exposure. However, there are several longitudinal studies, including a study of highly exposed workers,

which show decreased risks of asthma among PFOA-exposed workers. Furthermore, numerous factors have been shown to cause asthma as well as exacerbate symptoms, making it difficult to evaluate any potential relationship between PFASs exposure and risk of this condition. Host risk factors for asthma include a parental history of asthma, genetic polymorphisms, gender (higher preadolescent prevalence in boys; higher post-adolescent prevalence in girls), African-American or Puerto Rican Hispanic race/ethnicity, and obesity. Environmental risk factors include aeroallergen sensitization, respiratory viruses, cigarette smoke, air pollution, vitamin D deficiency, and stress. Microbial exposure in early life has been shown to reduce the risk of asthma, while prenatal exposure to tobacco smoke, stress, antibiotics, and emergency cesarean section delivery increase the risk of asthma in infants and children. Asthma may be triggered or exacerbated by house dust mite allergens, indoor or outdoor molds, pets, cockroaches, rodents, pollens, viral infections, active or passive smoking, air pollution, meteorological changes, dust, drugs, stress, and sulfite consumption. Collectively, the epidemiologic evidence does not support a conclusion that exposure to any PFAS, including PFOA or PFOS, increases the risk of developing asthma.

d. *Reproductive Effects:* Collectively, based on the currently available epidemiologic evidence, exposure to any PFAS, including PFOA and PFOS exposure, do not appear to be associated with preeclampsia, gestational hypertension, subfecundity, semen quality, miscarriage, puberty development, irregular menstrual cycles, menopause status, low birth weight, or adverse reproductive outcomes. While the potential relationship between exposure to different PFAS and reproductive outcomes has been evaluated in numerous studies, many of these studies are cross-sectional in design, or are longitudinal analyses of background exposure levels, which are insufficient to establish causation. Although several studies have reported associations between PFASs exposure and adverse reproductive effects, many associations are not statistically significant, results vary widely within and between studies, innumerable factors (that are commonly uncontrolled for) influence as well as modify reproductive outcomes, and the potential clinical relevance for many results are of undetermined implication. In addition, the currently available epidemiologic evidence for some factors, such as menstrual cycles and timing of menopause, are limited by data from relatively few studies. Findings from these studies may be attributable to reverse causation.

e. *Immunotoxicity and Autoimmunity:* The epidemiologic evidence does not support an independent relationship between exposure to any PFAS, including PFOA and PFOS exposure, and adverse immune function parameters. This conclusion is made on the following scientific basis: 1) the currently available epidemiologic evidence originates from studies with widely varying levels of methodological quality with considerable data generated from cross-sectional studies that are not designed to evaluate temporal relationships, 2) inconsistent findings both within and between studies are reported, 3) the levels of most clinical findings are within the normal

range of the broader population, 4) laboratory values and serum measurements evaluated across studies are influenced and modified by numerous factors (e.g., medication use, dietary patterns, alcohol, genetics) that are not completely controlled for in the analyses, 5) clinical marker levels are examined that may be of undetermined clinical significance, and many studies of PFASs exposure have not shown increased risks for diseases or health-related states that are correlated with the clinical markers.

i. Immune Function: Overall, the currently available epidemiologic evidence on whether exposure to any PFAS may affect immune function is inconsistent. Markers of immune function vary widely across the literature, thus, limiting the ability to make comparisons between studies. Even among studies measuring the same markers of immune function, differences in the study populations and timing of exposure measures to PFAS make it challenging to interpret the results. Furthermore, these data are highly inconsistent among similar immune outcomes with some studies finding positive associations, some finding inverse associations, some finding no associations, and others finding associations only among subgroups of the population. The large number of cross-sectional studies in this section further limit the ability to accurately and reliably interpret these positive, inverse and null findings. Methodological differences mentioned above related to study design, study populations, windows of exposure, age at outcomes evaluation and potential for outcome misclassification may explain some of the inconsistency in findings.
ii. Vaccinations: Taken together, the evidence pertaining to exposure to any PFAS, including PFOA and PFOS exposure, and vaccine response originates from heterogeneous adult and child study populations. While some analyses within and between studies indicate inverse associations between PFOA or PFOS exposure and antibody response, most associations are not statistically significant. In addition, there is some inconsistency in study results by various factors, such as gender and vaccination. For example, some studies observe the strongest inverse associations with tetanus while others observe the strongest association with diphtheria. Furthermore, several of the birth cohort studies were conducted among a population from the Faroe Islands, which limits the generalizability of these data.
iii. Infections: Collectively, the data from prospective birth cohort studies indicate some variable positive associations between PFOA and PFOS exposure and some infections. However, the data are not consistent across studies, and all studies have important methodological limitations. Thus, the currently available epidemiologic evidence does not support a conclusion that exposure to any PFAS is independently associated with increasing the risk of infections or infectious diseases among pediatric or adult populations.

iv. Allergy: Data from the prospective birth cohort studies do not support an association between exposure to any PFAS, including PFOA or PFOS exposure, and allergy outcomes including eczema or atopic dermatitis.
v. Ulcerative Colitis/Crohn's Disease: The epidemiologic evidence linking PFOA and/or PFOS exposure and ulcerative colitis is limited and is not sufficient to support a causal relationship. However, currently available data for Crohn's Disease, specifically, does not support any association with PFOA exposure.
vi. Rheumatoid Arthritis: The currently available epidemiologic evidence pertaining to PFASs exposure, namely PFOA exposure, and risk of rheumatoid arthritis is limited and not sufficient to draw a conclusion on any potential relationship.

f. *Other Health Parameters*:

i. Obesity: Collectively, the currently available epidemiologic evidence pertaining to exposure to any PFAS, including PFOA and PFOS exposure, and risk of overweight status or obesity is limited and inconsistent. Moreover, several studies that have addressed this topic have been cross-sectional analyses of study participants with background or near-background PFASs exposure levels.
ii. Duration of Breastfeeding: Data pertaining to PFASs exposure and duration of breastfeeding are sparse and inconsistent. Foremost among the methodological limitations across these studies is the limited adjustment for potentially relevant confounding factors, such as maternal breastfeeding education, smoking history, and dyad separation among other factors. Importantly breastfeeding is a source of PFAS elimination. As such, longer lactation may reduce PFAS serum levels, and explain the lower concentrations among those who breastfed longer.
iii. Sex Hormones: Collectively, exposure to PFOA or PFOS does not appear to be associated with – or impact – levels of testosterone, estradiol, or SHBG. Findings across studies have been inconsistent, and when differences have been reported, hormone levels have commonly been within normal ranges.
iv. Liver Function: Collectively, the epidemiologic evidence does not support a relationship between PFOA or PFOS exposure and adverse liver biomarker alterations, either individually, or as a composite group of liver function markers. Furthermore, the epidemiologic evidence does not support an increased risk between PFOA or PFOS exposure and adverse liver function or liver disease.
v. Kidney Function: Positive associations and/or correlations between PFAS, namely PFOA exposure, and uric acid levels have been reported in some studies. However, even in the presence of statistically significant differences between exposed and unexposed groups or between higher vs. lower levels of exposure, the magnitude

of the uric acid level difference between study groups is minimal and values remain within the normal range. Thus, findings from these studies are not supportive of an adverse effect of exposure to any PFAS on uric acid levels.

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Dr. Alexander, PhD, MSPH, has extensive experience in health research methodology and disease causation assessments, particularly in the conceptualization, design, analysis, and interpretation of epidemiologic studies. He is a leading expert in meta-analysis methodology. He has published on a diverse range of topics and types of studies, including original epidemiologic research, qualitative reviews, systematic weight-of-evidence assessments, and quantitative meta-analyses. Because of his expertise in research methodology, Dr. Alexander has served as principal investigator on numerous projects involving a wide variety of exposures and health outcomes. His research areas include, but are not limited to: occupational and environmental exposures, such as asbestos, benzene, solvents, pesticides, and perfluorinated compounds; community health studies and cluster investigations involving air, water, and soil exposures; clinical, pharmacoepidemiology, and medical device studies including clinical trial data evaluations. In addition, Dr. Alexander has extensive experience in nutritional epidemiology and has conducted systematic reviews and meta-analyses of dietary and nutritional factors and cancer, cardiovascular disease, type 2 diabetes, hypertension, and body composition. His work in this area has involved studies of dietary patterns, intake of whole foods, and dietary supplements, such as meat and fat intake, dairy and egg consumption, breakfast eating, multivitamin and mineral supplements, fish oil, caffeine, coffee, and infant formula.

Dr. Alexander has 210 peer-reviewed manuscripts, professional presentations, published abstracts, and book chapters. He frequently presents on the understanding and interpretation of epidemiologic evidence in a variety of professional venues, such as national conventions, scientific conferences, and governmental regulatory forums. Dr. Alexander serves on the editorial boards of the American Journal of Clinical Nutrition, PLOS ONE, and Frontiers in Nutrition Methodology. In addition, he has served on scientific committees and scientific advisory groups. Dr. Alexander was awarded a National Cancer Institute Fellowship for Cancer Prevention and Control to complete his doctorate in epidemiology, and was the 2010 recipient of the UAB School of Public Health alumnus award for scientific excellence, based on recognition of his "significant scientific contributions through demonstrated commitment and exemplary leadership in empirical research, research methodology, or theory building or adaptation." Dr. Alexander is a former Visiting Professor of Epidemiology at the University of Copenhagen in Copenhagen, Denmark, and is currently an Affiliate Associate Professor at Colorado State University.

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Education

2004	Ph.D., Epidemiology, University of Alabama-Birmingham School of Public Health
	Concentration in cancer epidemiology
2001	M.S.P.H., Epidemiology and Biostatistics, University of South Florida College of Public Health
	Concentration in analytical methodology
1997	B.A.S., Community Public Health, University of Minnesota
	Minors in chemistry, mathematics, and psychology

Employment

2019-Present	President and Principal Epidemiologist, MetaMethod, South Lyon, Michigan
2014–2018	Principal Epidemiologist and Office Director, EpidStat Institute, Ann Arbor, Michigan
2004–2014	Principal Epidemiologist, Exponent Inc. Health Sciences, Chicago, Illinois and Boulder, Colorado
2001–2004	Research Assistant, University of Alabama-Birmingham, Department of Epidemiology and Department of Pathology, National Cancer Institute Cancer Prevention and Control Fellowship, Birmingham, Alabama
2000–2001	Research Assistant, Moffitt Cancer Center, Department of Radiology, Digital Medical Imaging Program, Tampa, Florida
2000–2001	Teaching Assistant, Advanced Epidemiology Methods, Department of Epidemiology and Biostatistics, University of South Florida

Academic Appointments and Responsibilities

2020	Internship Preceptor, University of Wisconsin, Criminal Justice/Legal Studies program
2019-present	Affiliate Associate Professor, Department of Animal Sciences, Colorado State University
2018	Internship Preceptor, University of Michigan, Department of Epidemiology

2018	Epidemiology Department Internship Poster Session, Nov. 2, 2018; University of Michigan Department of Public Health
2016–2019	Visiting Professor, Department of Nutrition, Exercise and Sports, Faculty of Science, University of Copenhagen, Denmark

Honors and Awards

2020	Mayo Clinic Proceedings *Highly Cited Author* award for 2020: *A Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies of Eicosapentaenoic and Docosahexaenoic Long-Chain Omega-3 Fatty Acids and Coronary Heart Disease Risk* [Second Award]
2019	Mayo Clinic Proceedings *Highly Cited Author* award for 2019: *A Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies of Eicosapentaenoic and Docosahexaenoic Long-Chain Omega-3 Fatty Acids and Coronary Heart Disease Risk*
2019	PLOS ONE: Top 10% most cited author 2015
2018	Appointed to the editorial board of PLOS ONE
2017	Most read article 2016: *Meta-analysis of egg consumption and risk of coronary heart disease and stroke*; Journal of the American College of Nutrition
2016	Most read article 2015: *Red Meat and Colorectal Cancer: A Quantitative Update on the State of the Epidemiologic Science*; Journal of the American College of Nutrition
2015	Appointed to the editorial board of the American Journal of Clinical Nutrition
2013	Certificate of Achievement, Decker Communication Training
2010	UAB School of Public Health Alumnus Award for Scientific Excellence
2010	MDLinx Featured Article
2001–2004	National Cancer Institute Cancer Fellowship, Cancer Prevention and Control Training Program, University of Alabama-Birmingham
2003	William C. Bailey Award for Excellence in Cancer Prevention and Control Research, UAB Comprehensive Cancer Center Annual Research Retreat
2002	Lifetime Member of MENSA High Intelligence Society
2000–2001	Academic Fellowship, University of South Florida

Professional Organizations

2009–Present	American Society of Nutrition (ASN)
2005–Present	Society for Epidemiologic Research (SER)
2003–Present	American College of Epidemiology (ACE)

2011–2013	International Society of Pharmacoepidemiology (ISPE)
2005–2008	International Society for Environmental Epidemiology (ISEE)
2005–2008	American Public Health Association (APHA)
1999–2001	Infectious Disease Association (IDSA)

Primary Areas of Expertise

Meta-analysis methodology

Systematic reviews and weight-of-evidence assessments

Disease causation assessments

Occupational and environmental epidemiology

Nutritional epidemiology

Community health studies and alleged cluster evaluations

Clinical trial support

Chronic diseases, including cancer, cardiovascular disease, and type 2 diabetes

Dietary and lifestyle factors, such as food and supplement intake, smoking behaviors, body weight, and physical activity

Public speaking with a focus on interpreting and articulating epidemiologic evidence

Editorial Boards

2018-Present	Editorial Board, *PLOS ONE*
2017	Guest Editor, *Nutrients*
2015-Present	Editorial Board, *American Journal of Clinical Nutrition*
2014–Present	Associate Editor, *Frontiers in Nutrition Methodology*

Peer Reviewer (Abridged List)

American Journal of Clinical Nutrition

American Journal of Epidemiology

Epidemiology

Journal of the National Cancer Institute

Annals of Oncology

Regulatory Toxicology and Pharmacology

Nutrition and Cancer

Public Health Nutrition

Journal of Food Composition and Analysis

Risk Assessment

Cancer

Cancer Epidemiology Biomarkers and Prevention

American Journal of Preventive Medicine

European Journal of Cancer Prevention

Obesity

Southern Medical Journal

International Journal of Cancer

Journal of Women's Health

British Journal of Cancer

Peer-Reviewed Publications

1. Bylsma LC, Dean R, Lowe K, Sangaré L, Alexander DD, Fryzek JP. The incidence of infusion reactions associated with monoclonal antibody drugs targeting the epidermal growth factor receptor in metastatic colorectal cancer patients: A systematic literature review and meta-analysis of patient and study characteristics. Cancer Med. 2019 Aug 3. doi: 10.1002/cam4.2413.
2. Cohen SS, Alexander DD, Krebs NF, Young BE, Cabana MD, Erdmann P, Hays NP, Bezold C, Levin-Sparenberg E, Turini M, Saavedra J. Factors Associated with breastfeeding initiation and continuation: A Meta-Analysis. J Pediatr. 2018 Dec;203:190-196.e21
3. Fryzek JP, Reichert H, Summers N, Townes L, Deuson R, Alexander DD, Vanderpuye-Orgle J. Indirect treatment comparison of cabazitaxel for patients with metastatic castrate-resistant prostate cancer who have been previously treated with a docetaxel-containing regimen. PLoS One. 2018 Apr 11;13(4):e0195790.
4. Alexander DD. In Reply I - Prescribing More Stringent Design of Randomized Clinical Trials of Omega-3 Polyunsaturated Fatty Acids. Mayo Clinic Proceedings 2017 Jun;92(6):1006-1007.
5. Alexander DD, Miller PE, van Elswyk M, Kuratko C, Bylsma L. A Meta-Analysis of Randomized Controlled Trials and Prospective Cohort Studies of Eicosapentaenoic and Docosahexaenoic Long Chain Omega-3 Fatty Acids and Coronary Heart Disease Risk. Mayo Clinic Proceedings 2017 Jan;92(1):15-29.
6. Alexander DD, Miller PE, Vargas A, Weed DL, Cohen SS. Meta-analysis of egg consumption and risk of coronary heart disease and stroke. J Am Coll Nutr. 2016 Nov-Dec;35(8): 704-716.

7. Alexander DD, Yan J, Bylsma LC, Northington RS, Grathwohl D, Steenhout P, Erdmann P, Spivey-Krobath E, Haschke F. Growth of infants consuming whey-predominant term infant formulas with a protein content of 1.8 g/100 kcal: a multicenter pooled analysis of individual participant data. Am J Clin Nutr. 2016 Oct;104(4):1083-1092.
8. Maki KC, Guyton JR, Orringer CE, Hamilton-Craig I, Alexander DD, Davidson MH. Triglyceride-lowering therapies reduce cardiovascular disease event risk in subjects with hypertriglyceridemia. J Clin Lipidol. 2016 Jul-Aug;10(4):905-14.
9. Garabrant DH, Alexander DD, Miller PE, Fryzek JP, Boffetta P, Teta MJ, Hessel PA, Craven VA, Kelsh MA, Goodman M. Response to Kay Teschke. Re: Mesothelioma among Motor Vehicle Mechanics: An Updated Review and Meta-analysis. Ann Occup Hyg. 2016 Oct;60(8):1036-7.
10. Alexander DD, Bylsma LC, Elkayam L, Nguyen DL. Nutritional and health benefits of semi-elemental diets: A comprehensive summary of the literature. World J Gastrointest Pharmacol Ther. 2016 May 6;7(2):306-19.
11. J Fryzek, D Alexander, N Summers, J Fraysse, H Reichert, L Townes, J Vanderpuye-Orgle. Indirect Treatment Comparison Of Cabazitaxel For Patients With Metastatic Castration-Resistant Prostate Cancer Who Have Been Previously Treated With A Docetaxel-Containing Regimen. Value in Health. 2016 May 19(3): A139-A140.
12. Alexander DD, Weed DL. On the need for improved methodologic quality of published reviews. Am J Clin Nutr. 2016 Mar;103(3):683-4.
13. Alexander DD, Bylsma LC, Vargas AJ, Cohen SS, Doucette A, Mohamed M, Irvin SR, Miller PE, Watson H, Fryzek JP. Dairy Consumption and Cardiovascular Disease: A Systematic Review and Meta-Analysis. Br J Nutr. 2016 Feb;115(4):737-50.
14. Garabrant DH, Alexander DD, Miller PE, Fryzek JP, Boffetta P, Teta MJ, Hessel PA, Craven VA, Kelsh MA, Goodman M. Mesothelioma among Motor Vehicle Mechanics: An Updated Review and Meta-analysis. Ann Occup Hyg. 2016 Jan;60(1):8-26.
15. Weaver CM, Alexander DD, Boushey CJ, Dawson-Hughes B, Lappe JM, LeBoff MS, Liu S, Looker AC, Wallace TC, Wang DD. Calcium plus vitamin D supplementation and risk of fractures: an updated meta-analysis from the National Osteoporosis Foundation. Osteoporos Int. 2016 Jan;27(1):367-76.
16. Bylsma LC, Alexander DD. A review and meta-analysis of prospective studies of red and processed meat, meat cooking methods, heme iron, heterocyclic amines and prostate cancer. Nutr J. 2015 Dec 21;14(1):125.
17. Alexander DD, Weed DL, Miller PE, Mohamed MA. 2015. Red Meat and Colorectal Cancer: A Quantitative Update on the State of the Epidemiologic Science, J Am Coll Nutr. 2015 Nov-Dec;34(6):521-43
18. Alexander DD, Bassett JK, Weed DL, Barrett EC, Watson H, Harris W. Meta-Analysis of Long-Chain Omega-3 Polyunsaturated Fatty Acids (LCω-3PUFA) and prostate cancer, Nutr Cancer. 2015;67(4):543-54
19. Yurko-Mauro K, Alexander DD, Van Elswyk ME. 2015. Docosahexaenoic Acid and Adult Memory: A Systematic Review and Meta-Analysis. PLoS One. 2015 Mar 18;10(3).

20. Alexander DD, Jiang X, Bylsma LC, Garabrant DH, Irvin SR, Fryzek JP. Historical cancer incidence and mortality assessment in an Illinois community proximal to a former manufactured gas plant. BMJ Open. 2014 Dec 22;4(12).

21. Veruva SY, Steinbeck MJ, Toth J, Alexander DD, Kurtz SM. 2014. Which Design and Biomaterial Factors Affect Clinical Wear Performance of Total Disc Replacements? A Systematic Review. Clin Orthop Relat Res. 2014 Dec;472(12):3759-69

22. Tsuji JS, Perez V, Garry MR, Alexander DD. 2014. Association of low-level arsenic exposure in drinking water with cardiovascular disease: A systematic review and risk assessment. Toxicology 323:78-94.

23. Tsuji JS, Alexander DD, Perez V, Mink PJ. 2014. Arsenic exposure and bladder cancer: quantitative assessment of studies in human populations to detect risks at low doses. Toxicology 317:17-30.

24. Miller PE, Alexander DD, Perez V. 2014. Effects of whey protein and resistance exercise on body composition: a meta-analysis of randomized controlled trials. J Am Coll Nutr 33:163-175.

25. Schmier JK, Miller PE, Levine JA, Perez V, Maki KC, Rains TM, Devareddy L, Sanders LM, Alexander DD. 2014. Cost savings of reduced constipation rates attributed to increased dietary fiber intakes: a decision-analytic model. BMC Public Health 14:374. doi: 10.1186/1471-2458-14-374.:374-14.

26. Miller PE, Van EM, Alexander DD. 2014. Long-chain omega-3 fatty acids eicosapentaenoic acid and docosahexaenoic acid and blood pressure: a meta-analysis of randomized controlled trials. Am J Hypertens 27:885-896.

27. Miller PE, Alexander DD, Weed DL. 2014. Uncertainty of Results in Nutritional Epidemiology. Nutrition Today 49:147-152.

28. Alexander DD. 2013. No association between meat intake and mortality in Asian countries. Am J Clin Nutr 98:865-866.

29. Huhmann MB, Perez V, Alexander DD, Thomas DR. 2013. A self-completed nutrition screening tool for community-dwelling older adults with high reliability: a comparison study. J Nutr Health Aging 17:339-344.

30. Goswami E, Craven V, Dahlstrom DL, Alexander DD, Mowat F. 2013. Domestic asbestos exposure: a review of epidemiologic and exposure data. Int J Environ Res Public Health 10:5629-5670.

31. Alexander DD, Bailey WH, Perez V, Mitchell ME, Su S. 2013. Air ions and respiratory function outcomes: a comprehensive review. J Negat Results Biomed 12:14. doi: 10.1186/1477-5751-12-14.:14-12.

32. Perez V, Alexander DD, Bailey WH. 2013. Air ions and mood outcomes: a review and meta-analysis. BMC Psychiatry 13:29. doi: 10.1186/1471-244X-13-29.:29-13.

33. Alexander DD, Weed DL, Chang ET, Miller PE, Mohamed MA, Elkayam L. 2013. A systematic review of multivitamin-multimineral use and cardiovascular disease and cancer incidence and total mortality. J Am Coll Nutr 32:339-354.

34. Maki KC, Van Elswyk ME, Alexander DD, Rains TM, Sohn EL, McNeill S. 2012. A meta-analysis of randomized controlled trials that compare the lipid effects of beef versus poultry and/or fish consumption. J Clin Lipidol 6:352-361.

35. Kanas GP, Taylor A, Primrose JN, Langeberg WJ, Kelsh MA, Mowat FS, Alexander DD, Choti MA, Poston G. 2012. Survival after liver resection in metastatic colorectal cancer: review and meta-analysis of prognostic factors. Clin Epidemiol 4:283-301.

36. Holscher HD, Czerkies LA, Cekola P, Litov R, Benbow M, Santema S, Alexander DD, Perez V, Sun S, Saavedra JM, Tappenden KA. 2012. Bifidobacterium lactis Bb12 enhances intestinal antibody response in formula-fed infants: a randomized, double-blind, controlled trial. JPEN J Parenter Enteral Nutr 36:106S-117S.

37. Bryan NS, Alexander DD, Coughlin JR, Milkowski AL, Boffetta P. 2012. Ingested nitrate and nitrite and stomach cancer risk: an updated review. Food Chem Toxicol 50:3646-3665.

38. Alexander DD, Weed DL, Mink PJ, Mitchell ME. 2012. A weight-of-evidence review of colorectal cancer in pesticide applicators: the agricultural health study and other epidemiologic studies. Int Arch Occup Environ Health 85:715-745.

39. Alexander DD, Weed DL, Cushing CA, Lowe KA. 2011. Meta-analysis of prospective studies of red meat consumption and colorectal cancer. Eur J Cancer Prev 20:293-307.

40. Alexander DD, Cushing CA. 2011. Red meat and colorectal cancer: a critical summary of prospective epidemiologic studies. Obes Rev 12:e472-e493.

41. Kelsh MA, Alexander DD, Mink PJ, Mandel JH. 2010. Occupational trichloroethylene exposure and kidney cancer: a meta-analysis. Epidemiology 21:95-102.

42. Moolgavkar SH, Turim J, Alexander DD, Lau EC, Cushing CA. 2010. Potency factors for risk assessment at Libby, Montana. Risk Anal 30:1240-1248.

43. Anderson B, Hardin JM, Alexander DD, Grizzle WE, Meleth S, Manne U. 2010. Comparison of the predictive qualities of three prognostic models of colorectal cancer. Front Biosci (Elite Ed) 2:849-56.:849-856.

44. Alexander DD, Wagner ME. 2010. Benzene exposure and non-Hodgkin lymphoma: a meta-analysis of epidemiologic studies. J Occup Environ Med 52:169-189.

45. Alexander DD, Schmitt DF, Tran NL, Barraj LM, Cushing CA. 2010. Partially hydrolyzed 100% whey protein infant formula and atopic dermatitis risk reduction: a systematic review of the literature. Nutr Rev 68:232-245.

46. Alexander DD, Morimoto LM, Mink PJ, Lowe KA. 2010. Summary and meta-analysis of prospective studies of animal fat intake and breast cancer. Nutr Res Rev 23:169-179.

47. Alexander DD, Miller AJ, Cushing CA, Lowe KA. 2010. Processed meat and colorectal cancer: a quantitative review of prospective epidemiologic studies. Eur J Cancer Prev 19:328-341.

48. Alexander DD, Mink PJ, Cushing CA, Sceurman B. 2010. A review and meta-analysis of prospective studies of red and processed meat intake and prostate cancer. Nutr J 9:50. doi: 10.1186/1475-2891-9-50.:50-59.

49. Alexander DD, Cabana MD. 2010. Partially hydrolyzed 100% whey protein infant formula and reduced risk of atopic dermatitis: a meta-analysis. J Pediatr Gastroenterol Nutr 50:422-430.

50. Erdreich LS, Alexander DD, Wagner ME, Reinemann D. 2009. Meta-analysis of stray voltage on dairy cattle. J Dairy Sci 92:5951-5963.

51. Alexander DD, Cushing CA. 2009. Quantitative assessment of red meat or processed meat consumption and kidney cancer. Cancer Detect Prev 32:340-351.

52. Alexander DD, Cushing CA, Lowe KA, Sceurman B, Roberts MA. 2009. Meta-analysis of animal fat or animal protein intake and colorectal cancer. Am J Clin Nutr 89:1402-1409.

53. Mink PJ, Alexander DD, Barraj LM, Kelsh MA, Tsuji JS. 2008. Low-level arsenic exposure in drinking water and bladder cancer: a review and meta-analysis. Regul Toxicol Pharmacol 52:299-310.

54. Mandel JH, Kelsh M, Mink PJ, Alexander DD. 2008. Trichloroethylene exposure and non-Hodgkin's lymphoma: supportive evidence (letter). Occup Environ Med 65:147-148.

55. Kelsh MA, Alexander DD, Kalmes RM, Buffler PA. 2008. Personal use of hair dyes and risk of bladder cancer: a meta-analysis of epidemiologic data. Cancer Causes Control 19:549-558.

56. Alexander DD. 2007. An environmental cause of orofacial cleft defects or an unexplained cluster? South Med J 100:553-554.

57. Alexander DD, Waterbor J, Hughes T, Funkhouser E, Grizzle W, Manne U. 2007. African-American and Caucasian disparities in colorectal cancer mortality and survival by data source: an epidemiologic review. Cancer Biomark 3:301-313.

58. Alexander DD, Mink PJ, Adami H-O, Cole P, Mandel JS, Oken MM, Trichopoulos D. 2007. Multiple myeloma: A review of the epidemiologic literature. Int J Cancer 120:40-46.

59. Alexander DD, Mink PJ, Adami HO, Chang ET, Cole P, Mandel JS, Trichopoulos D. 2007. The non-Hodgkin lymphomas: a review of the epidemiologic literature. Int J Cancer 120 Suppl 12:1-39.

60. Alexander DD, Kelsh MA, Mink PJ, Mandel JH, Basu R, Weingart M. 2007. A meta-analysis of occupational trichloroethylene exposure and liver cancer. Int Arch Occup Environ Health 81:127-143.

61. Mandel JH, Kelsh MA, Mink PJ, Alexander DD, Kalmes RM, Weingart M, Yost L, Goodman M. 2006. Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review. Occup Environ Med 63:597-607.

62. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. 2006. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukaemia. Occup Med (Lond) 56:485-493.

63. Chatla C, Jhala NC, Katkoori VR, Alexander D, Meleth S, Grizzle WE, Manne U. 2005. Recurrence and survival predictive value of phenotypic expression of Bcl-2 varies with tumor stage of colorectal adenocarcinoma. Cancer Biomark 1:241-250.

64. Saif MW, Alexander D, Wicox CM. 2005. Serum Alkaline Phosphatase Level as a Prognostic Tool in Colorectal Cancer: A Study of 105 patients. J Appl Res 5:88-95.

65. Alexander DD, Jhala N, Chatla C, Steinhauer J, Funkhouser E, Coffey CS, Grizzle WE, Manne U. 2005. High-grade tumor differentiation is an indicator of poor prognosis in African Americans with colonic adenocarcinomas. Cancer 103:2163-2170.
66. Alexander DD, Chatla C, Funkhouser E, Meleth S, Grizzle WE, Manne U. 2004. Postsurgical disparity in survival between African Americans and Caucasians with colonic adenocarcinoma. Cancer 101:66-76.
67. Manne U, Alexander DD, Chatla C. 2004. Author Reply: Postsurgical Disparity in Survival between African Americans and Caucasians with Colonic Adenocarcinoma. Cancer 101:2900.

Scientific Presentations and Speaking Engagements

1. Alexander DD. Theory: Bias and Confounding. NIH Funded Short-Course: Strengthening Causal Inference in Behavioral Obesity Research. Module 2 Observational Studies: Advantages, Limits, and Best Practices Indiana University. June 8 – 12, 2020 (postponed).
2. Alexander DD. Theory: Bias and Confounding. NIH Funded Short-Course: Strengthening Causal Inference in Behavioral Obesity Research. Indiana University. July 29-August 2, 2019.
3. Alexander DD. The Epidemiology of Talc-Related Disease. DRI, Austin, TX. November 9, 2018.
4. Alexander DD. Eating less red meat: the evidence behind the recommendation. IFT2018: A Matter of Science + Food, Chicago, IL. July 17, 2018.
5. Alexander DD. The diet connection to cancer: understanding how food can cause, prevent or impact cancer risk. FoodFluence, Lisbon, Portugal. January 12-16, 2018.
6. Alexander DD. Epidemiology of red meat and cancer. Health Canada, Ottawa, Canada. December 15, 2017.
7. Alexander DD. Interpreting the epidemiology of red meant and cancer. International Meat Secretariat (IMS) Human Nutrition and Health Committee meeting in Paris, France, October 3, 2017.
8. Alexander DD. Epidemiology of egg consumption and risk of coronary heart disease and stroke. Webinar: What does the science say? Eggs and heart health. Egg Farmers of Canada. June 6, 2017
9. Alexander DD. Consumption: diet and lifestyle perspective. Meat and Livestock Australia Scientific Workshop. Sydney, Australia. April 10, 2017.
10. Alexander DD. Are red meat consumers unhealthy? Nutrition in Action Symposium. Sydney, Australia. April 5, 2017.
11. Alexander DD. Red meat and cancer risk: interpreting the evidence. NCBA Discovery Symposium. Denver, CO. July 27, 2016.
12. Alexander DD. Theory: bias and confounding. Strengthening Causal Inference in Behavioral Obesity Research. NIH Funded Short-Course; University of Alabama-Birmingham, July 25, 2016.

13. Alexander DD. Red meat and cancer risk: interpreting the evidence. Danish Nutrition Society; University of Copenhagen. Copenhagen, Denmark. June 21, 2016.
14. Alexander DD. Meta-analysis: recycling garbage or an important tool for evaluating the evidence? Drug and Medical Device Seminar. Chicago, IL. May 19-20, 2016.
15. Alexander DD. Evaluating the relationship of meat and cancer risk. Canadian Nutrition Society, Ottawa, Canada. May 5-7, 2016.
16. Alexander DD. Becoming a nutrition detective. Washington State Academy of Nutrition and Dietetics, Annual Conference. Vancouver, WA. April 18, 2016.
17. Alexander DD. Red meat and chronic disease: A closer look into the data. Utah Academy of Nutrition and Dietetics, Annual Conference. Ogden, UT. March 24, 2016.
18. Alexander DD. Meat and cancer risk: understanding the science. Protein: Contributions and Controversies. Toronto, Canada. February 29, 2016.
19. Alexander DD. Understanding the role of epidemiology in disease causation. Asbestos Medicine; DRI. Las Vegas, NV, November 5-6, 2015.
20. Alexander DD. Theory: bias and confounding in observational studies. Strengthening Causal Inference in Behavioral Obesity Research. NIH Funded Short-Course; University of Alabama-Birmingham, July 20, 2015.
21. Alexander DD. Red and processed meat consumption and cancer. International Meat Society. Calgary, Canada. July 1-2, 2015.
22. Alexander DD. Red meat consumption and chronic disease. Canadian Nutrition Society. Winnipeg, Canada. May 30, 2015.
23. Alexander DD. Understanding studies of diet and chronic disease. New Mexico Academy of Nutrition and Dietetics. Albuquerque, NM. April 24, 2015.
24. Alexander DD. Overview of FDA Health Claims and the Submission Process. Webinar. January 13, 2015.
25. Alexander DD. Becoming a Nutrition Detective: Critically Reviewing Research and Communicating Science. DBC Communications Camp, Academy of Nutrition and Dietetics. Las Vegas, NV; January 17, 2015.
26. Alexander DD, State of the epidemiologic science on red meat and chronic disease. Health Canada. Ottawa, Canada; October 22, 2014
27. Alexander DD. Observational epidemiologic studies of breakfast intake. Kellogg Scientific Advisory Board Meeting. Battle Creek, MI; October 1, 2014.
28. Alexander DD. Caffeine intake during pregnancy: the pregnancy signal and reproductive outcomes. The Toxicology Forum, 40th Annual Summer Meeting, Aspen, CO, July 7-10, 2014.
29. Alexander DD. Understanding studies of diet and chronic disease. Delaware Dietetic Association, Dover, DE, May 9, 2014.
30. Alexander DD. Red meat and colorectal cancer: a quantitative update on the state of the science. Experimental Biology, San Diego, CA, April 27, 2014.

31. Alexander DD. Nutrition Detective: An Epidemiologist's Investigation into Diet and Chronic Disease. 31st Annual Health & Nutritional Sciences Conference, South Dakota State University, April 10, 2014.
32. Alexander DD. Summarizing, Interpreting, and Communicating Epidemiologic Evidence. GOED Exchange, Salt Lake City, UT, February 6, 2014.
33. Alexander DD. Synthesizing and Summarizing Epidemiology Evidence, Health Economics, and Fiber and Constipation. Food & Fiber Summit: Identifying Practical Solutions to Meet America's Fiber Needs, Washington DC, January 28, 2014.
34. Alexander DD. Interpreting Epidemiologic Evidence, and a Case Study on Red Meat and Colorectal Cancer. Oncology Nutrition Symposium, Hollywood, FL, January 18, 2014.
35. Alexander DD. OMEGA-3 LC-PUFAs: Judging the Epidemiologic Evidence. GOED Fall Member Meeting at the SupplySide West Tradeshow, Las Vegas, NV, November 14, 2013.
36. Alexander DD. Nutritional Epidemiology: Are We Overstating the Evidence? Missouri Academy of Family Physicians, 21stAnnual Fall Conference, Branson, MO, November 9, 2013
37. Alexander DD. Interpreting Epidemiologic Evidence. DRI Asbestos Medicine Seminar, New Orleans, LA, November 8, 2013.
38. Alexander DD. DRI Research Roundtable: Full-Fat Dairy Products in Nutrition and Health (panel discussant). October 10, 2013.
39. Alexander DD. Update on Red Meat and Colorectal Cancer. International Meat Society Annual Meeting. Granada Spain (webinar), September 14, 2013.
40. Alexander DD. Sustainable Nutrition Roundtable (panel discussant). August 2, 2013.
41. Alexander DD. Dairy and body composition: Making sense of meta-analyses. Dairy Research Institute Meeting: Dairy and Weight, Chicago, IL, June 4–5, 2013.
42. Alexander DD. Nitrate and nitrite exposure and stomach cancer: summary of the epidemiologic evidence. Canadian Nutrition Society, Annual Meeting, Quebec City, Canada, May 31, 2013.
43. Alexander DD. Meta-analysis: Judging the evidence, fish oil and cardiovascular disease. AOCS: Omega-3 Fatty Acids and Heart Health, Montreal, Canada, April 28–May 1, 2013.
44. Alexander DD. Epidemiologic evidence: Investigation Into diet and chronic disease. MINK Conference: Nutrition Without Boundaries, Kansas City, KS, April 6, 2013.
45. Alexander DD. A systematic review of multivitamin use and mortality, cardiovascular disease, and cancer. Council for Responsible Nutrition (CRN): Day of Science, Laguna Beach, CA, October 2–3, 2012.
46. Alexander DD. Diet and cancer: Are we asking the right question? Cancer Society of New Zealand, New Zealand Ministry of Health, Network Communications, Wellington, New Zealand, September 11, 2012.

47. Alexander DD. Interpreting meta-analyses for dietetic practice. Professional development session for New Zealand dietitians, University of Otago, Dunedin, New Zealand, September 10, 2012.

48. Huhmann MB, Kaspar KM, Perez V, Alexander DD, Thomas DR. Accuracy of a new self-completed nutrition screening tool for community-dwelling older adults. Oral Presentation at the European Society for Clinical Nutrition and Metabolism, Barcelona, Spain, September 8–11, 2012.

49. Alexander DD. Interpreting meta-analysis for dieticians in practice. International Congress of Dietetics, Dieticians Association of Australia. Sydney, Australia, September 7, 2012.

50. Alexander DD. Red meat and colorectal cancer: Are we asking the right question(s)? Diet and Gut Health Symposium. Nutrition Society of Australia. Sydney, Australia, September 5, 2012.

51. Alexander DD. Diet and gut health round table meeting and presentation. Meat & Livestock Australia, Sydney, Australia, September 4, 2012.

52. Alexander DD. An update on red meat and cancer. Webinar, International Congress of Meat Science and Technology, Montreal, Canada, August 12–17, 2012.

53. Alexander DD. How to improve the research integrity of meta-analyses and systematic reviews. Scientific Approaches to Strengthening Research Integrity in Nutrition and Energetics, Mohonk Mountain House, NY, August 7–8, 2012.

54. Alexander DD. Sustainable agriculture and the integration of plant- and animal-based foods. California Milk Advisory Board, San Francisco, CA, July 25, 2012.

55. Alexander DD. Nitrate and nitrite exposure and stomach cancer: Summary of the epidemiologic evidence. IFT Annual Meeting, Las Vegas, NV, June 25–28, 2012.

56. Perez V, Schmier JK, Alexander DD. Race/ethnic disparities in pediatric discharges from all US community, non-rehabilitation hospitals for respiratory syncytial virus (RSV) among children one year or younger. Oral presentation at the 45th Annual Society for Epidemiologic Research (SER) Meeting, Minneapolis, MN, June 27–30, 2012.

57. Alexander DD. The nutrition detective: An epidemiologist's look at diet and chronic disease conundrums. New York State Dietetic Association 2012 Annual Meeting & Expo, Albany, NY, May 4–5, 2012.

58. Alexander DD. Epidemiology: Methods for weighing the evidence. MDLA Young Lawyers Meeting, Minneapolis, MN, February 9, 2012.

59. Alexander DD. Nutritional epidemiology: Weighing the evidence and a case study on red meat intake and colorectal cancer. MeatEat Nutritional Conference, Oslo, Norway, September 1, 2011.

60. Alexander DD. Prevalence of bone metastasis from breast, lung or prostate cancer: A systematic and quantitative review of the literature. International Conference on Pharmacoepidemiology, Chicago, IL, August 15–17, 2011.

61. Alexander DD. Benzene epidemiology: Weighing the evidence and a case study of non-Hodgkin lymphoma. Benzene Litigation Conference Audiocast, Chicago, IL, July 13, 2011.

62. Alexander DD. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.

63. Alexander DD. Translating the science: Red meat & cancer. Ranch Event 2011, Texas Beef Council, San Antonio, TX, June 2, 2011.

64. Alexander DD. Epidemiology consulting and a case study on red meat and cancer. Distinguished Alumni Investigator Lecture, Birmingham, AL, March 23, 2011.

65. Alexander DD. Nutritional epidemiology: Weighing the evidence. International Life Sciences Institute-ILSI North America Annual Meeting, Orlando, FL, January 24–25, 2011.

66. Alexander DD. The nutrition detective: Translating nutrition science into practice. Texas Dietetics Association. December 8, 2010 (webinar).

67. Alexander DD. The epidemiology of red and processed meat consumption and cancer and cardiovascular disease. The role of red meats in a healthy diet: U.S. Meat Export Federation, Mexico City, Mexico, October 20, 2010 (Keynote speaker).

68. Alexander DD. Meat consumption and cancer: An epidemiologic overview. Live Well, Napa Valley, June 10, 2010.

69. Alexander DD. Red meat consumption and colorectal cancer: A meta-analysis of prospective studies. Experimental Biology, Anaheim, CA, April 26, 2010.

70. Alexander DD. A weight-of-evidence review of colorectal cancer in pesticide applicators: The Agricultural Health Study and other Epidemiologic Studies. CropLife America/Rise Spring Conference, Washington DC, April 15, 2010.

71. Alexander DD. Meat and Cancer. American Meat Institute, Spring Meeting, April 14, 2010.

72. Alexander DD, Weed DL. Ongoing assessment of pesticides and colorectal cancer: A weight of evidence evaluation of epidemiologic literature. Environmental Protection Agency SAP draft framework, Washington DC, February 2, 2010.

73. Alexander DD. Benzene exposure and non-Hodgkin lymphoma: a meta-analysis. Society for Epidemiologic Research, Anaheim, CA, June 24, 2009 (Spotlight Session).

74. Alexander DD. The epidemiology of red and processed meat and cancer. IMS Human Nutrition and Health Committee meeting, Chicago, IL, May 20, 2009 (Invited Speaker).

75. Erdreich LS, Wagner M, Van Kerkhove M, Alexander DD. Stray voltage meta-analysis: needs, methods and challenges. 46th Annual Rural Energy Conference, La Crosse, WI, February 28, 2008.

76. Alexander DD. Epidemiologic evaluation of red meat and cancer. Cattle Industry Convention & Trade Show, Nutrition Roundtable, Reno, NV, February 7, 2008.

77. Alexander DD. Red meat scientific assessment. Industry Stakeholder Cancer Forum, Chicago, IL, October 11, 2007.

78. Alexander DD. Meta-analysis of occupational trichloroethylene exposure and lymphohematopoietic malignancies and liver cancer. Epidemiology Seminar Series, University of Illinois, Chicago, IL, November 17, 2006.

79. Kelsh MA, Mandel JH, Mink PJ, Weingart M, Alexander DD, Goodman M. A meta-analysis of kidney cancer, non-Hodgkin's lymphoma and occupational trichloroethylene exposure. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.

80. Mink PJ, Alexander DD, Barraj L, Kelsh Ma, Tsuji J. A review and meta-analysis of low-level arsenic exposure in drinking water and bladder cancer. Presentation to the Canadian Pest Management Regulatory Agency, Ottawa, Canada, June 2006.

81. Mandel JH, Alexander DD, Kelsh MA. Occupational trichloroethylene exposure: recent insights from epidemiologic and toxicologic perspectives. State of the Art Conference of the American College of Occupational and Environmental Medicine, Chicago, IL, October 2005.

Book Chapter

1. Kelsh MA, Alexander DD. Occupational and environmental epidemiology. In: Encyclopedia of Epidemiology. Sage Publications, Thousand Oaks, CA, 2007.

Published Abstracts

1. Lowe K, Bylsma L, Dean R, Gillezeau C, Sangare L, Alexander DD, Fryzek J. The incidence of infusion reactions associated with monoclonal antibody drugs targeting the epidermal growth factor receptor in metastatic colorectal cancer patients: A systematic literature review and meta-analysis. American Society for Clinical Oncology, 2019 Gastrointestinal Cancers Symposium; J Clin Oncol 37, 2019 (suppl 4; abstr 526).

2. Lowe K, Bylsma L, Levin-Sparenberg L, Sangare L, Fryzek J, Alexander DD. Prevalence of *KRAS*, *NRAS*, and *BRAF* gene mutations in metastatic colorectal cancer patients: A systematic literature review and meta-analysis. American Society for Clinical Oncology, 2019 Gastrointestinal Cancers Symposium; J Clin Oncol 37, 2019 (suppl 4; abstr 523).

3. Bylsma L, Alexander DD. A Review and Meta-Analysis of Prospective Studies of Red and Processed Meat, Meat Cooking Methods, Heme Iron, Heterocyclic Amines and Prostate Cancer. Experimental Biology, San Diego, CA, April 2-6, 2016.

4. Miller PE, Alexander DD. A Review and Meta-Analysis of Prospective Studies of Red and Processed Meat and Pancreatic Cancer. Experimental Biology, San Diego, CA, April 2-6, 2016.

5. Althuis M, Alexander DD, Frankenfeld F, Weed DL. Meta-analysis of observational studies in context: sugar-sweetened beverages and type 2 diabetes. Federation of American Societies for Experimental Biology (FASEB). March, 2015

6. Alexander DD, Weed DL. Red meat and colorectal cancer: a quantitative update on the state of the science. Experimental Biology, San Diego, CA, April 26-30, 2014.

7. Pyatt, D and Alexander, D. A meta-analysis of AML subtypes reported in cigarette Smokers. The Bone Marrow Niche, Stem Cells, and Leukemia: Impact of Drugs, Chemicals, and the Environment, May 29-31, New York Academy of Sciences. 2013.

8. Alexander DD, Mitchell M, Taylor A, Lowe K, Langeberg W, et al. Prevalence of bone metastasis in breast cancer patients and subsequent survival: A systematic and quantitative review of the literature. San Antonio Breast Cancer Symposium, San Antonio, TX, December 6–10, 2011.
9. Mitchell M, Taylor A, Lowe K, Langeberg W, Alexander DD, et al. Prevalence of bone metastasis from breast, lung or prostate cancer: A systematic and quantitative review of the literature. International Conference on Pharmacoepidemiology, Chicago, IL, August 15–17, 2011.
10. Taylor A, Kanas G, Primrose J, Langeberg W, Alexander DD, et al. Survival after surgical resection of hepatic metastases from colorectal cancer: An updated review and meta-analysis. World Congress on Gastrointestinal Cancer, Barcelona, Spain, June 22–25, 2011.
11. Alexander DD, Perez V, Cushing C, Weed DL. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.
12. Perez V, Alexander DD, Cushing C. Processed meat consumption and stomach cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.
13. Maki KC, Van Elswyk ME, Alexander DD, Rains TM, Sohn EL, McNeill S. A meta-analysis of randomized controlled trials comparing lipid effects of beef with poultry and/or fish consumption. National Lipid Association Annual Scientific Sessions, May, 2011; Abstract 393.
14. Alexander DD. Meta-analysis of prospective epidemiologic studies of red meat intake and colorectal cancer. American Association for Cancer Research, Orlando, FL, April 2–6, 2011.
15. Alexander DD, Cabana MD. Partially hydrolyzed 100% whey protein infant formula and reduced risk of atopic dermatitis: A meta-analysis. Pediatric Academic Societies, Vancouver, BC, Canada, May 1–4, 2010.
16. Alexander DD, Cushing CA. A meta-analysis of red or processed meat intake and prostate cancer. Society for Epidemiologic Research, Anaheim, CA, 2009.
17. Alexander DD, Wagner ME, Kelsh MA. Benzene exposure and non-Hodgkin lymphoma: A meta-analysis. Society for Epidemiologic Research, Anaheim, CA, June 24, 2009.
18. Alexander DD, Schmitt D, Tran N, Barraj L, Cushing CA. Partially hydrolyzed 100% whey infant formula and atopic dermatitis risk reduction: A systematic review of the literature. Experimental Biology, New Orleans, LA, 2009.
19. Alexander DD, Cushing CA, Lowe KL. Meta-analysis of animal fat intake and colorectal cancer. Experimental Biology, New Orleans, LA, 2009.
20. Alexander DD, Cushing CA, Roberts MA. Quantitative assessment of red and processed meat intake and kidney cancer. Experimental Biology, New Orleans, LA, 2009.
21. Lowe KL, Alexander DD, Morimoto LM. Meta-analysis of animal fat intake and breast cancer. Experimental Biology, New Orleans, LA, 2009.
22. Morimoto LM, Alexander DD, Cushing CA. Meta-analysis of red and processed meat consumption and breast cancer. Experimental Biology, New Orleans, LA, 2009.

23. Manne U, Grizzle WE, Alexander DD, Katkoori V. Racial differences in colorectal cancer: the need to educate clinicians and researchers for improved patient care. American Association for Cancer Education, 41st Annual Meeting, Birmingham, AL, October 2007.
24. Gatto NM, Alexander DD, Kelsh MA. A meta-analysis of occupational exposure to hexavalent chromium and stomach cancer. Epidemiology, Sept 2007; Vol 18, issue 5, pS33.
25. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukemia. Proceedings 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.
26. Kelsh MA, Mandel JH, Mink PJ, Weingart M, Alexander DD, Goodman M. A meta-analysis of kidney cancer, non-Hodgkin's lymphoma and occupational trichloroethylene exposure. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.
27. Mink PJ, Alexander DD, Barraj L, Kelsh MA, Tsuji J. Meta-analysis of low level arsenic exposure and bladder cancer. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.
28. Alexander DD, Mink PJ, Butchko H. How "fast food" is used and interpreted in scientific research: methodological considerations. Proceedings, Experimental Biology 2006, San Francisco, CA, April 2006.
29. Kapica CM, Alexander DD, Mink PJ, Butchko H. The definition of fast food in published studies. Proceedings, Experimental Biology 2006, San Francisco, CA, April 2006.
30. Alexander D, Chatla C, Funkhouser E, Jhala N, Grizzle WE, Manne U. Racial differences in survival based on tumor differentiation and stage in patients who have undergone surgery for colon cancer. J Clin Oncol 2004; 22:14S (July Supplement).
31. Alexander D, Funkhouser E, Saif M. Alkaline phosphatase (AP) as a prognostic tool in colorectal cancer (CRC). Proceedings, American Society of Clinical Oncology 2003; 22:354.
32. Malhotra P, Kallergi M, Alexander D, et al. Discrepancies between film and digital mammography interpretations. Medical Imaging 2002, Proceedings of SPIE (The International Society for Optical Engineering), February 2002.
33. Alexander D, Malhotra P, Kallergi M, et al. Digital vs. film mammography: calcification interpretation. American Association of Physicists in Medicine 2001, (July Supplement).

Poster Presentations at Scientific Conferences

1. Bylsma L, Levin-Sparenberg L, Lowe K, Sangare L, Fryzek J, Alexander DD. Prevalence of *KRAS*, *NRAS*, and *BRAF* gene mutations in metastatic colorectal cancer patients: A systematic literature review and meta-analysis. International Society for Pharmacoepidemiology; Spotlight Molecular Epidemiology, Biomarkers, and Pharmacogenetics; Philadelphia, PA, August 28, 2019.
2. Bylsma L, Dean R, Lowe K, Gillezeau C, Sangare L, Alexander DD, Fryzek J. The incidence of infusion reactions associated with monoclonal antibody drugs targeting the epidermal growth factor receptor in metastatic colorectal cancer patients: A systematic literature review and

meta-analysis. International Society for Pharmacoepidemiology; Biologics/Biosimilars; Philadelphia, PA, August 28, 2019.

3. Lowe K, Bylsma L, Dean R, Gillezeau C, Sangare L, Alexander DD, Fryzek J. The incidence of infusion reactions associated with monoclonal antibody drugs targeting the epidermal growth factor receptor in metastatic colorectal cancer patients: A systematic literature review and meta-analysis. American Society for Clinical Oncology, 2019 Gastrointestinal Cancers Symposium; San Francisco, CA, January 19, 2019.
4. Lowe K, Bylsma L, Levin-Sparenberg L, Sangare L, Fryzek J, Alexander DD. Prevalence of *KRAS*, *NRAS*, and *BRAF* gene mutations in metastatic colorectal cancer patients: A systematic literature review and meta-analysis. American Society for Clinical Oncology, 2019 Gastrointestinal Cancers Symposium; San Francisco, CA, January 19, 2019.
5. Yaser J, Alexander DD. Processed meat consumption and risk of colorectal cancer: a systematic review and meta-analysis. Epidemiology Department Internship Poster Session, University of Michigan Department of Public Health; Ann Arbor, MI, Nov. 2, 2018.
6. Weed DL, Alexander DD. A systematic review of Hill's criteria. Society of Toxicology. San Antonio, TX, March 11-15, 2018.
7. Fryzek J, Alexander DD, Summers N, Fraysse J, Reichert H, Townes L, Vanderpuye-Orgle J. Indirect treatment comparison of cabazitaxel for patients with metastatic castration-resistant prostate cancer who have been previously treated with a docetaxel-containing regimen. International Society for Pharmacoeconomics and Outcomes Research (ISPOR). Washington DC. May 21-25, 2016
8. Miller PE, Alexander DD. A Review and Meta-analysis of Prospective Studies of Red and Processed Meat and Pancreatic Cancer. Experimental Biology. San Diego, CA. April 4, 2016.
9. Maki KC, Guyton JR, Orringer CE, Hamilton-Craig I, Alexander DD, Davidson MH: Triglyceride-lowering therapies reduce cardiovascular disease event risk in subjects with hypertriglyceridemia. National Lipid Association Scientific Sessions, Chicago, IL, USA, June 11–14, 2015
10. Bylsma L, Miller P, Alexander DD. Meta-analysis of red meat intake and type 2 diabetes. Experimental Biology. Boston, MA, March 31, 2015.
11. Tsuji JS, Alexander DD, Perez V. Low-level arsenic in drinking water and bladder cancer risk: Meta-analysis update and risk assessment implications. Annual Meeting of the Society of Toxicology, San Antonio, TX, March 10–14, 2013.
12. Perez V, Alexander DD, Bailey WH. Air ions and mood outcomes: A review and meta-analysis. Poster presentation at the American College of Epidemiology, Chicago, IL, September 8–11, 2012.
13. Huhmann MB, Kaspar KM, Perez V, Alexander DD, Thomas DR. Accuracy of a self-completed nutrition screening tool for community-dwelling older adults when completed by the patient or caregivers. Poster presentation at the International Academy on Nutrition and Aging Meeting, Albuquerque, NM, July 12–13, 2012.

14. Perez V, Schmier JK, Alexander DD. Race/ethnic disparities in pediatric discharges from all US community, non-rehabilitation hospitals for respiratory syncytial virus (RSV) among children one year or younger. Oral presentation at the 45th Annual Society for Epidemiologic Research (SER) Meeting, Minneapolis, MN, June 27–30, 2012.
15. Alexander DD, Mitchell M, Taylor A, Lowe K, Langeberg W, et al. Prevalence of bone metastasis in breast cancer patients and subsequent survival: A systematic and quantitative review of the literature. San Antonio Breast Cancer Symposium, San Antonio, TX, December 6–10, 2011.
16. Alexander DD, Perez V, Cushing C, Weed DL. Red meat consumption and colorectal cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.
17. Perez V, Alexander DD, Cushing C. Processed meat consumption and stomach cancer: A meta-analysis of prospective epidemiologic studies. Congress of Epidemiology, Montreal, Canada, June 21, 2011.
18. Gatto NM, Alexander DD, Kelsh MA. A meta-analysis of occupational exposure to hexavalent chromium and stomach cancer. 19th Annual International Society of Environmental Epidemiology Conference, Mexico City, Mexico, September 5–9, 2007.
19. Alexander DD, Mink PJ, Mandel JH, Kelsh MA. A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukemia. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.
20. Mink PJ, Alexander DD, Barraj L, Kelsh MA, Tsuji J. Meta-analysis of low level arsenic exposure and bladder cancer. Proceedings, 2nd North American Congress of Epidemiology, Seattle, WA, June 2006.
21. Alexander DD, Mink PJ, Butchko H. How "fast food" is used and interpreted in scientific research: methodological considerations. Experimental Biology 2006, San Francisco, CA, April 2006.
22. Kapica CM, Alexander DD, Mink PJ, Butchko H. The definition of fast food in published studies. Experimental Biology 2006, San Francisco, CA, April 2006.
23. Mink PJ, Alexander DD, Barraj LM, Kelsh MA, Tsuji JS. Meta-analysis of low level arsenic exposure and bladder cancer: implications for risk assessment in the United States. 45th Annual Meeting of the Society of Toxicology, San Diego, CA, March, 2006.
24. Alexander D, Jhala N, Chatla C, Steinhauer J, Funkhouser E, Coffey C, Grizzle WE, Manne U. Racial differences in survival based on tumor differentiation and stage in patients who have undergone surgery for colon cancer. The 2004 American Society of Clinical Oncology Annual Meeting, New Orleans, LA, June 2004.
25. Chatla C, Alexander D, Manne U. Prognostic significance of Bcl-2 expression and p53 nuclear accumulation based on nodal status in patients with colorectal adenocarcinoma. The 95th Annual meeting of the American Association for Cancer Research, Orlando, Florida, March 2004.

26. Alexander D. Post-surgical disparity in survival between African-Americans and Caucasians with colonic adenocarcinomas. The UAB Comprehensive Cancer Center Annual Research Retreat, Birmingham, Alabama, October 2003.
27. Malhotra P, Kallergi M, Alexander D, et al. Discrepancies between film and digital mammography interpretations. The annual meeting for Medical Imaging: Observer Performance Studies, San Diego, CA, February 26–28, 2002. (Poster Presentation, Presenter: P. Malhotra).
28. Alexander D, Malhotra P, Kallergi M, et al. Digital vs. film mammography. The American Association of Physicists in Medicine Conference, Salt Lake City, UT, July 22–26, 2001.

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Legal testimony of Dominik D. Alexander, PhD, MSPH, December 7, 2016 – December 7, 2020

Date	Case Name	Jurisdiction	Activity
12/16/16	Kenneth Vigneron vs. E.I. du Pont de Nemours and Co.	United States District Court, Southern District of Ohio, Eastern Division	Trial testimony
1/24/17	Roderick Currie and Marie Sundling-Currie vs. Borg Warner Corporation, et al.	Superior Court of the State of California, County of Los Angeles	Deposition
1/26/17	James W. Coates and Kareen Coates, Plaintiffs, vs. Bennett Auto Supply, et al.	17th Judicial Circuit Court, Broward County, Florida	Deposition
2/8/17	Larry O. Moody vs. E.I. du Pont de Nemours and Co.	United States District Court, Southern District of Ohio, Eastern Division	Trial testimony
2/10/17	Manual Castillo and Hilda Castillo vs. 3M Company, et al.	California Superior Court, Alameda County	Deposition
2/14/17	James W. Coates and Kareen Coates, Plaintiffs, vs. Bennett Auto Supply, et al.	17th Judicial Circuit Court, Broward County, Florida	Trial testimony
2/17/17	Donald Stone vs. A.W. Chesterton Company et al.	King County Superior Court, Washington	Deposition
3/16/17	Roderick Currie and Marie Sundling-Currie vs. Borg Warner Corporation, et al.	Superior Court of the State of California, County of Los Angeles	Trial testimony
3/24/17	Brian and Lena Bell vs. AC&R Insulation Co., Inc.	Superior Court of the District of Columbia	Deposition
5/5/17	Linda Granere and Deborah Mains, Individually and as Co-Personal Representatives of the Estate of Robert Granere vs. 84 Lumber Co., et al.	17th Judicial Circuit Court, Broward County, Florida	Deposition
6/7/17	Council for Education and Research on Toxics vs. Starbucks Corporation, a Washington corporation; et al.	Superior Court of the State of California, County of Los Angeles, Central Civil West	Deposition
6/8/17	Joyce Moore vs. Borg Warner Corporation	17th Judicial Circuit Court, Broward County, Florida	Deposition
6/9/17	Charles Kenyon vs. 3M Company, et al.	Superior Court of the State of California, County of San Diego	Deposition

Date	Case Name	Jurisdiction	Activity
7/26/17	Varon V. Philyaw and Joyce Bowen Philyaw vs. 3M Company, et al.	United States District Court, South Carolina, Anderson Division	Deposition
7/27/17	Ernest J. Foucha vs. Aims Group, Inc., et al.	Civil District Court for the Parish of Orleans, Louisiana	Deposition
7/28/17	James C. Sizemore vs. Reilly-Benton Company, Inc., et al.	Civil District Court for the Parish of Orleans, Louisiana	Deposition
8/9/17	Stacey Thomas vs. Monster Beverage Corporation, a Delaware Corporation, et al.	Superior Court of the State of California, County Riverside	Deposition
8/11/17	Sharon Styron, Individually and as Special Administrator for the Estate of Bryan Rutledge, vs. PACCAR, et al.	Third Judicial Circuit Court, Madison County, Illinois	Deposition
9/6/17-9/7/17	Council for Education and Research on Toxics vs. Starbucks Corporation, a Washington corporation; et al.	Superior Court of the State of California, County of Los Angeles, Central Civil West	Trial testimony
9/22/17	Bonita Williams, Individually and as Successor-in-Interest to Decedent Roger Williams; David Williams; Michael Williams; and Steven Williams vs. Advance Stores Company, Inc., et al.	Superior Court of the State of California for the County of Los Angeles	Deposition
9/26/17	John G. Serrano and Rachel Serrano vs. All American Home Center, Inc., et al.	Superior Court of the State of California County of Alameda	Deposition
9/28/17	Katherine M. Uyeno v. The Old Oahu Tug Service, Inc., et al.	Circuit Court of the First Circuit, Hawaii	Deposition
10/05/17	Larry Petrie and Charlotte Petrie v. John Crane Inc., et al.	Superior Court of California, County of Solano	Deposition
10/12/17	Rodney Stevenson, et al. vs. MCIC, Inc., et al.	Circuit Court for Washington County (Baltimore), Maryland	Deposition
10/18/17	Ronald Seals vs. Air & Liquidated System, et al.	Superior Court of the State of California County of Alameda	Deposition
10/24/17	Mary Klukkert v. Bird Inc., et al.	13th Judicial Circuit Court, Hillsborough County, Florida	Deposition
10/26/17, 2/5/18	Enrique Dominguez and Olma Dominguez vs. Autozone West, Inc., et al.	Superior Court of the State of California for the County of Los Angeles	Deposition
12/21/17	Kathleen McCarthy-Brooke vs. Air & Liquid Systems Corporation, et al.	Superior Court of the State of Washington for the county of King	Deposition
1/5/18	Eldon Angle, et al. vs. American Lumber Company, et al	Superior Court of the State of California for the County of Los Angeles	Deposition

Date	Case Name	Jurisdiction	Activity
2/12/18	Max Rosenfield and Maria Rosenfield, his wife vs. A.W. Chesterton Company, et al.	Third Judicial Circuit Court, Madison County, Illinois	Deposition
2/13/18	Steven Sober and Glenn Sober, individually and as successor-in-interest to Vera Sober, Deceased vs. CIL&D, LLC, et al.	Superior Court of the State of California for the County of Los Angeles	Deposition
3/5/18	William Albert Hawkins, et al. vs. BorgWarner Morse TEC, et al.	Circuit Court for Prince George County, Maryland	Deposition
3/12/18	Michael B. Donohue and Anne Donohue, his wife vs. ABB, Inc., et al.	Supreme Court of the State of New York for the County of New York	Deposition
3/13/18	Kenneth Kramer vs. 3M Company, et al.	Superior Court of the State of California for the County of Alameda	Deposition
4/11/18	Victor Michel vs. Ford Motor Company, et al.	Civil District Court for the Parish of Orleans, Louisiana	Deposition
5/10/18	Ronald Blake, et al. vs. ACandS, Inc., et al.	Circuit Court for Baltimore City, Maryland	Deposition
6/6/18	Susan C. Whitmire vs. Ingersoll Rand Company, et al.	7th Judicial Court, Spartanburg County, South Carolina	Deposition
6/7/18	Donald Knutson, et al. vs. Air & Liquid Systems Corporations, et al.	Superior Court of the State of California for the County of Alameda	Deposition
6/8/18	Leslie Jack, et al. vs. Asbestos Corporation, et al.	US District Court, Western District of Washington at Seattle	Deposition
6/21/18	Charles and Gayle Ross vs. Appleton GRP, LLC, et al.	Superior Court of the State of Indiana for the County of Marion	Deposition
6/29/18	Ron M. Long, Sr. and Sheila Long vs. BorgWarner Morse TEC, Inc., et al.	Superior Court of the State of Georgia for the County of Catoosa	Deposition
8/2/18	Joanna M. Summerlin vs. Philip Morris USA, et al.	Superior Court of the Commonwealth of Massachusetts for the County of Middlesex	Deposition
8/10/18	Karen Bearden, et al. vs. DAP Products, Inc., et al.	California Superior Court, Los Angeles County	Deposition
8/15/18	John C. Dugger, Jr., et al. vs. Air & Liquid Systems Corporation, et al.	US District Court for the District of Maryland	Deposition
8/28/18	Barbara Barr vs. A-1 Clutch Co., et al.	California Superior Court, Alameda County	Deposition
8/30/18	James and Carol Burton, and Dale Erwin vs. ACH Food Companies Inc., et al.	Eleventh Judicial Circuit Court, McLean County, Illinois	Deposition
8/30/18	Norman Phillips vs. Air & Liquid Systems Corporation, et al.	Eleventh Judicial Circuit Court, McLean County, Illinois	Deposition
8/30/18	Doris Thomas vs. Pneumo Abex LLC, et al.	Eleventh Judicial Circuit Court, McLean County, Illinois	Deposition

Date	Case Name	Jurisdiction	Activity
8/31/18	Melissa Hale, et al. vs. Ford Motor Company, et al.	California Superior Court, Solano County	Deposition
9/10/18	Steven E. Bolin and Deborah Bolin vs. Bakers Pride Oven Company, LLC, et al.	17th Judicial Circuit Court, Broward County, Florida	Deposition
10/2/18, 10/3/18	Cody Dean Bledsoe vs. Monster Beverage Corporation, et al.	California Superior Court, Riverside County	Deposition
10/22/18	Houshang Sabetian and Soraya Sabetian vs. Air & Liquid Systems Corporation, et la.	California Superior Court, Los Angeles County	Deposition
11/15/18	Pearl Tart, et al. vs. BorgWarner Morse TEC, LLC, et al.	California Superior Court, Los Angeles County	Deposition
11/26/18	Lakhota M. Harker vs. A.W. Chesterton Company, et al.	Third Judicial Circuit Court, Madison County, Illinois	Deposition
11/30/18	Depuy Orthopaedics Inc. Pinnacle Hip Implant Products Liability Litigation	US District Court, Northern District of Texas, Dallas Division	Deposition
12/5/18	Cody Dean Bledsoe v. Monster Beverage Corporation, et al.	California Superior Court, Riverside County	Trial testimony
12/20/18	Kelvin A. Hendrix v. Akebono Brake Corporation, et al.	Circuit Court of Saline County, Arkansas	Deposition
1/4/19	Rita Joyce Glenn vs. 3M Company, et al.	10th Judicial Circuit Court, Anderson County, South Carolina	Deposition
1/14/19	Viola Adams, et al. vs. Meritor, Inc., et al.	US District Court, Northern District of Mississippi, Greenville Division	Day of Science Testimony
1/17/19	Robert Scheurman and Marie Scheurman vs. Bird Corporation, et al.	13th Judicial Circuit Court, Hillsborough County, Florida	Deposition
1/18/19	Bonnie Leischner vs. AERCO International, Inc., et al.	11th Judicial Circuit Court, McLean County, Illinois	Deposition
1/22/19	Linda Wagner vs. Honeywell International, Inc., et al.	Division 11 Circuit Court, Jefferson County, Kentucky	Deposition
1/24/19	Elena David, et al. vs. BorgWarner Morse TEC LLC, et al.	California Superior Court, Los Angeles County	Deposition
1/25/19	Lionel Tamplain vs. Taylor-Seidenbach, Inc., et al.	Civil District Court for the Parish of Orleans, Louisiana	Deposition
1/28/19	Carolyn Walquist and Howard Walquist vs. Advanced Auto Parts, Inc., et al.	12th Judicial Circuit Court, St. Clair County, Illinois	Deposition
1/30/19	Wesley Shepherd and Iona Shepherd vs. Borg Warner Morse TEC LLC, et al.	11th Judicial Circuit Court, McLean County, Illinois	Deposition
1/30/19	David Jones and Janet Jones vs. Bechtel Corporation, et al.	11th Judicial Circuit Court, McLean County, Illinois	Deposition
2/5/19	George H. Hogue and Frances G. Hogue vs. Allied Packing & Supply, Inc., et al.	California Superior Court, Alameda County	Deposition

Date	Case Name	Jurisdiction	Activity
2/6/19	Anthony J. Licciardi, III vs. Taylor-Seidenbach, Inc., et al.	Civil District Court for the Parish of Orleans, Louisiana	Deposition
2/27/19	Mary Ann Corder vs. 3M Company, et al.	California Superior Court, Los Angeles County	Deposition
2/28/19	Barbara Maddy vs. Honeywell International, Inc., et al.	Court of Common Pleas of Cuyahoga County, Ohio	Deposition
3/7/19	Houshang Sabetian, et al. vs. Air & Liquid Systems Corporation, et al.	California Superior Court, Los Angeles County	Trial testimony
3/28/19	Barbara Collyer vs. Honeywell International, Inc., et al.	Circuit Court of Crittenden County, Kentucky	Deposition
4/3/19	Jamie Conner and William Janke vs. 3M Corporation, et al.	New Jersey Superior Court, Middlesex County	Deposition
4/19/19	Joe W. Rucker and Gwendolyn Baptist-Rucker vs. Banner Supply Co., et al.	11th Judicial Circuit Court, Miami-Dade County, Florida	Deposition
4/25/19	Charles French and Victoria French vs. ArvinMeritor, Inc., et al.	11th Judicial Circuit Court, McLean County, Illinois	Deposition
5/2/19	Randolph Morton and Edna S. Morton vs. 3M Company, et al.	California Superior Court, Los Angeles County	Deposition
5/9/19	Charles E. Thornton and Constance Thornton vs. GEA Mechanical Equipment US, Inc., et al.	11th Judicial Circuit Court, Miami-Dade County, Florida	Deposition
6/10/19	Charles E. Thornton and Constance Thornton vs. GEA Mechanical Equipment US, Inc., et al.	11th Judicial Circuit Court, Miami-Dade County, Florida	Trial testimony
6/17/19	George Crudge vs. Amcord Inc., et al.	California Superior Court, Los Angeles County	Deposition
6/28/19	Julian Montoya and Lynn Ann Montoya vs. 3 Company, et al.	California Superior Court, Los Angeles County	Deposition
7/10/19	Sebastian Bretado vs. 3M Company, et al.	California Superior Court, Los Angeles County	Deposition
7/12/19 7/15/19	Randolph Morton and Edna S. Morton vs. 3M Company, et al.	California Superior Court, Los Angeles County	Trial testimony
7/16/19	Arthur Putt and Janet Putt vs. CBS Corporation, et al. (Part 1)	Los Angeles Superior Court	Deposition
7/18/19	Jeannie McCrystal and Mark McCrystal vs. Autozone, Inc. et al.	Commonwealth of Kentucky, Daviess Circuit Court	Deposition
7/19/19	Alfred Bennett and Pamela Bennett vs. General Gasket Corporation, et al.	State of Missouri, Twenty-Second Judicial Circuit (City of St Louis)	Deposition
7/23/19	Arthur Putt and Janet Putt vs. CBS Corporation, et al. (Part 2)	Los Angeles Superior Court	Deposition
8/20/19	Alfred Bennett and Pamela Bennett vs. General Gasket Corporation, et al.	State of Missouri, Twenty-Second Judicial Circuit (City of St Louis)	Trial testimony

Date	Case Name	Jurisdiction	Activity
8/21-8/22/19	Arthur Putt and Janet Putt vs. CBS Corporation, et al. (Part 2)	Los Angeles Superior Court	Trial testimony
8/26/19	Michael Charles Bell et al. vs. BorgWarner Morse Tec, Inc., et al.	Baltimore City Circuit Court	Deposition
8/26/19	Lloyd and Marilyn Hutton vs. American President Lines, Ltd., et al.	Los Angeles Superior Court	Deposition
8/27/19	Janet Finch (for Estate of William M. Finch) vs. BorgWarner Morse Tec LLC et al.	Baltimore City Circuit Court	Deposition
8/28/19	Larry Cole, Sr. (for the Estate of Larry Nathaniel Cole) et al. vs. Honeywell International Inc., et al.	Morgan County, Missouri Circuit Court	Deposition
8/29/19	Carol Steinberg (for the Estate of Terry Steinberg) vs. 3M Company et al.	Cook County, Illinois Circuit Court	Deposition
9/3/19	Diane Shoemaker French (for the Estate of Bobby Shoemaker) vs. Bostik, Inc. et al	17th Judicial Circuit Court, Broward County, FL	Deposition
9/10/19	Linda and Bruce Guill vs. America's Trailer Company LLC et al	California Superior Court, County of Los Angeles	Deposition
9/13/19	Margaret Jones (for the Estate of William Jones) vs. Akebono Brake Corp. et al.	Crawford County, Arkansas Circuit Court	Deposition
9/16/19	Richard Pifer (deceased) et al. vs. Barrett's Minerals et al.	Baltimore City Circuit Court	Deposition
9/23/19	Martin Boyer vs. 3M Co. et al.	22nd Judicial Circuit Court (St. Louis MO)	Deposition
10/1/19	Julian and Lynn Ann Montoya vs. 3M Company et al.	California Superior Court, County of Los Angeles	Deposition
10/7/19	Maryann Hudak (for the Estate of Richard Hudak) vs. Honeywell International, Inc. et al.	Court of Common Pleas, Cuyahoga County, OH	Deposition
10/14/19	Phillip Posey (for the Estate of Peggy Posey) vs. Honeywell International, Inc. et al.	Oklahoma County Superior Court	Deposition
10/29/19	Theresa Lynn Anderson for the Estate of Eugene G. Hohlfeld vs. Automotive Parts Headquarters of Eau Clair, Inc., et al.	La Crosse County Circuit Court, Wisconsin	Deposition
10/30/19	David and Francisca Lara vs. Agco Corporation et al.	Superior Court of California, Alameda County	Deposition
11/6/19	Maryann Hudak for the Estate of Richard C. Hudak vs. Honeywell International, Inc. et al.	Court of Common Pleas, Cuyahoga County, OH	Trial testimony

Date	Case Name	Jurisdiction	Activity
11/25/19	Larry D. Wille for the Estate of Treva Wille vs. Authorized Motor Parts Corp., et al	Cass County, Missouri, Seventeenth Judicial Circuit Court	Deposition
11/26/19	Steve J. Lusty vs. Norfolk Southern Railway Company & Consolidated Rail Corporation	Third Judicial Circuit Court, Wayne County, Michigan	Deposition
12/6/19	James H. Woodard, Jr. vs. A.W. Chesterton Co., et al.	Circuit Court of Cook County, Illinois	Deposition
12/9/19	Ron M. & Sheila Long vs. Borg Warner Morse TEC, Inc., et al	Superior Court of Catoosa County, Georgia	Daubert Hearing
12/12/19	Roy Hicks vs. Air & Liquid Systems Corporation et al.	Eleventh Judicial Circuit Court, McClean County, Illinois	Deposition
12/16/19	Robert N. Levesque for the Estate of Anne J. Levesque et al. vs. Colgate-Palmolive Company et al.	Alameda County Superior Court, California	Deposition
12/17/19	George and Valerie Lewis and Mike Shanahan vs. Air & Liquid Systems, Inc. et al.	11th Judicial Circuit Court, McLean County, Illinois	Deposition
1/7/20	Lloyd and Beverley Glieden vs. Honeywell International Inc., et al.	Alameda County Superior Court, California	Deposition
1/8/20	Paul H. and Mary M. Fankhauser vs. BorgWarner Morse TEC LLC et al.	Iowa District Court, Polk County	Deposition
1/9/20	Bruce and Janice Rhyne vs. US Steel Corporation et al.	US District Court for the Western District of North Carolina, Charlotte Division	Deposition
1/10/20	Willie McNeal, Jr. vs. Autozone Inc., et al.	Los Angeles County, Superior Court of California	Deposition
1/17/20	In re: Deepwater Horizon BELO Cases	United States District Court, Northern District of Florida, Pensacola Division	Deposition
1/27/20	Deborah Van Evera et al. vs. Asbestos Companies et al.	Alameda County, Superior Court, California	Deposition
1/28/20	Robert and Queida King vs. ABB, Inc. et al.	13th Judicial Circuit Court, Hillsborough County, Florida	Deposition
1/31/20	Inis and Gary Evans vs. 3M Company et al.	Kanawha County Circuit Court, West Virginia	Deposition
2/4/20	Reyna Carranza, individually and as successor-in-interest to Alberto Carranza (deceased) et al. vs. 3M Company et al.	Superior Court for Los Angeles, California	Deposition
2/5/20	Edward Chapman vs. Bennett Auto Supply, Inc, et al.	Circuit Court for Broward County, Florida	Deposition
2/11/20	Johnny and Sherry Pinkston vs. Air & Liquid Systems Corporation, et al.	17th Judicial Circuit Court, Broward County, Florida	Deposition
2/20/20	Calvin H. Evans III vs. Bartells Asbestos Settlement Trust et al.	Superior Court of Washington for Pierce County	Deposition

Date	Case Name	Jurisdiction	Activity
3/5/20	Barbara Ingram for the Estate of Samuel Ingram vs. American Honda Motor Co. Inc., et al.	Marion County Superior Court, Indiana	Deposition
3/6/20	Barbara Franklin, an individual; vs. Blue Bird Corporation, et al.	Superior Court for Los Angeles, California	Deposition
3/12/20	Jeffrey Richard Henry as Trustee doe next-of-kin of Richard Henry, dc'd vs. 3M Company, et al.	District Court for Ramsey, Minnesota	Trial testimony
3/17/20	Nikol Chuidian, et al. vs. AT&T Corp, et al.	Superior Court of California, County of Alameda	Deposition
3/20/20	Richard Kyler and Patricia Kyler vs. 3M Company, et al.	Superior Court of California, County of Los Angeles	Deposition
3/24/20	David Andrew Chaff, as representative of the estate of Ronald Chaff, et al. vs. AutoZone, Inc., et al.	Superior Court of Arizona, County of Maricopa	Deposition
3/27/20	Ricardo and Elvia Ocampo vs. AAMCO Transmissions, Inc. et al.	Superior Court of California, County of Alameda	Deposition
4/9/20	Connie Lee Fink and Robert Ray Fink vs. Air & Liquid Systems Corporation, et al.	Circuit Court of Milwaukee County, Wisconsin	Deposition
4/14/20	Karen Cornwell, Individually and as Administrator of the Estate of Roy Cork, (deceased) et al. vs. Marathon Petroleum Company LP.	District Court for the Southern District of Illinois	Deposition
4/21/20	Thomas H. Toy, Sr., and Agnes Toy vs. Honeywell International Inc., et al.	Superior Court of California, County of Alameda	Deposition
5/6/20	Melvin and Louise Butler vs. Cemex Construction Materials Florida, LLC, et al.	17th Judicial Circuit Court, Broward County, Florida	Deposition
5/13/20	Mellis Meyers and Judith Meyers vs. American Honda Motor Co. Inc., et al.	11th Judicial Circuit in and for Miami-Dade County, Florida	Deposition
6/17/20	George Sweikhart and Christina Sweikhart vs. Air & Liquid Systems Corporation, et al.	California Superior Court, Los Angeles County	Deposition
6/24/20	Cynthia Bowman and Vincent Bowman vs. Bayer Cropscience, Inc., et al.	1st Judicial Court, Orangeburg County, South Carolina	Deposition
7/22/20	William Maki v Metropolitan Life Insurance Company, et al.	Superior Court of the Commonwealth of Massachusetts for the County of Middlesex	Deposition
7/23/20	William Clark and Stephanie Clark vs. Arrow Machinery, Inc., et al.	King County Superior Court, Washington	Deposition
8/21/20	Steve and Cheryl Wiersema vs. 3M Company, et al.	Third Judicial Circuit Court, Madison County, Illinois	Deposition

Date	Case Name	Jurisdiction	Activity
8/26/20	Ricardo and Elvia Ocampo vs. AAMCO Transmissions, Inc. et al.	California Superior Court, Alameda County	Trial
9/3/20	Myrna Covarrubias Dena for the Estate of Daniel S. Dena et al. vs. Coty, Inc. et al.	California Superior Court, Alameda County	Deposition
9/15/20	Rosalino Reyes III and Gemma Reyes vs. Johnson & Johnson, et al.	California Superior Court, Alameda County	Deposition
9/22/20	Bruce Rhyne and Carol Rhyne vs. United States Steel Corporation, et al.	US District Court for the Western District of North Carolina, Charlotte Division	Trial
9/30/20	Steven Sarkis and Judy Sarkis vs. Advance Stores Company Incorporated, et al.	Superior Court of the Commonwealth of Massachusetts for the County of Middlesex	Deposition
10/5/20	James Robert McKeever vs. BP Exploration & Production Inc. & BP America Production Company	United States District Court Eastern District of Louisiana	Deposition
10/6/20	Joseph Derouen vs. ANCO Insulations Inc., et al.	19th Judicial District Court for the Parish of East Baton Rouge, State of Louisiana	Deposition
10/28/20	Carolyn and Nathaniel Wassinger vs. Air & Liquid Systems Corporation, et al.	California Superior Court, Los Angeles County	Deposition
11/2/20	Joe A. Coronado and Leticia Coronado vs. American Honda Motor Co., Inc., et al.	California Superior Court, Alameda County	Deposition
11/11/20	Judith Miller for the Estate of Freddy Miller, dec'd vs. ABB, Inc., et al.	Superior Court of Washington for Pierce County	Deposition
11/18/20	Michele Baker, et al. vs. Saint-Gobain Performance Plastics Corp., et al.	United States District Court, Northern District of New York	Deposition
11/19/20	Sharon Jennings for the Estate of Darrell Jennings, dec'd vs. Honeywell International, Inc., et al.	Court of Common Pleas of Cuyahoga County, Ohio	Deposition
11/24/20	Donald and Bertha Nadine West vs. A & A Key and Builders Supply, et al.	California Superior Court, Alameda County	Deposition

MetaMethod
South Lyon, MI 48178 ● 630-390-8190

MetaMethod Fee Schedule
Effective January 1, 2019

Professional Services (consultation, review of records, preparation of reports, telephone conversations, deposition and trial appearance, travel, meetings, etc.)

Dr. Dominik D. Alexander, Principal Epidemiologist……$425.00 per hour
Senior Scientist……...……..……...$250.00 per hour
Senior Epidemiologist …..……...$250.00 per hour
Senior Research Associate ……$225.00 per hour
Research Associate ……....$150.00 per hour
Epidemiologist….……..$150.00 per hour
Office Assistant…….. $80.00 per hour